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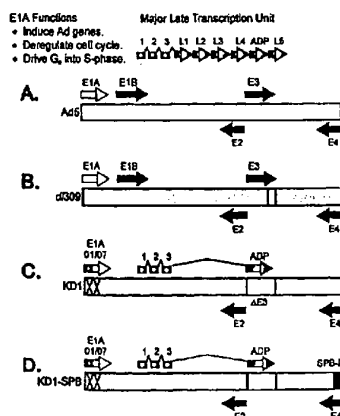
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(54) Title: REPLICATION-COMPETENT ANTI-CANCER VECTORS



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(57) Abstract: Novel vectors which are replication-competent in neoplastic cells and which overexpress an adenovirus death protein are disclosed. Some of the disclosed vectors are replication-restricted to neoplastic cells or to neoplastic alveolar type II cells. Compositions and methods for promoting the death of neoplastic cells using these replication-competent vectors are also disclosed.



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5 Background of the Invention

(1) Field of the Invention

This invention relates generally to the treatment of cancer and more particularly to vectors which replicate in neoplastic cells and which overexpress an adenovirus death protein (ADP) and to the use of these vectors in treating human cancer.

10 (2) Description of the Related Art

Cancer is a leading cause of death in the United States and elsewhere. Depending on the type of cancer, it is typically treated with surgery, chemotherapy, and/or radiation. These treatments often fail: surgery may not remove all the cancer; some cancers are resistant to chemotherapy and radiation therapy; and chemotherapy-resistant tumors frequently develop.

15 New therapies are necessary, to be used alone or in combination with classical techniques.

One potential therapy under active investigation is treating tumors with recombinant viral vectors expressing anti-cancer therapeutic proteins. Adenovirus-based vectors contain several characteristics that make them conceptually appealing for use in treating cancer, as well as for therapy of genetic disorders. Adenoviruses (hereinafter used interchangeably with

"Ads") can easily be grown in culture to high titer stocks that are stable. They have a broad host range, replicating in most human cancer cell types. Their genome can be manipulated by site-directed mutation and insertion of foreign genes expressed from foreign promoters.

- The adenovirion consists of a DNA-protein core within a protein capsid (reviewed by Stewart et al., "Adenovirus structure by x-ray crystallography and electron microscopy." in: *The Molecular Repertoire of Adenoviruses*, Doerfler, W. et al., (ed.), Springer-Verlag, Heidelberg, Germany, p. 25-38). Virions bind to a specific cellular receptor, are endocytosed, and the genome is extruded from endosomes and transported to the nucleus. The genome is a linear duplex DNA of about 36 kbp, encoding about 36 genes (Fig. 1A). In the nucleus, the "immediate early" E1A proteins are expressed initially, and these proteins induce expression of the "delayed early" proteins encoded by the E1B, E2, E3, and E4 transcription units (reviewed by Shenk, T., "Adenoviridae: the viruses and their replication" in: *Fields Virology*, Field, B.N. et al., Lippincott-Raven, Philadelphia, p. 2111-2148). E1A proteins also induce or repress cellular genes, resulting in stimulation of the cell cycle. About 23 early proteins function to usurp the cell and initiate viral DNA replication. Viral DNA replicates at about 7 h post-infection (p.i.), then late genes are expressed from the "major late" transcription unit. Major late mRNAs are synthesized from the common "major late promoter" by alternative pre-mRNA processing. Each late mRNA contains a common "tripartite leader" at its 5'-terminus (exons 1, 2, and 3 in Fig. 1), which allows for efficient translation of Ad late mRNAs. Cellular protein synthesis is shut off, and the cell becomes a factory for making viral proteins. Virions assemble in the nucleus at about 1 day p.i., and after 2-3 days the cell lyses and releases progeny virus. Cell lysis is mediated by the E3 11.6K protein, which has been renamed "adenovirus death protein" (ADP) (Tollefson et al., *J. Virol.* 70:2296-2306, 1996; Tollefson et al., *Virol.* 220:152-162, 1996). The term ADP as used herein in a generic sense refers collectively to ADP's from adenoviruses such as, e.g. Ad type 1 (Ad1), Ad type 2 (Ad2), Ad type 5 (Ad5) or Ad type 6 (Ad6) all of which express homologous ADP's with a high degree of sequence similarity.

- Human adenovirus type 5 (Ad5) is particularly useful for cancer gene therapy. It primarily causes asymptomatic or mild respiratory infections in young children, followed by long term effective immunity. Fatalities are extremely rare except when the patient is immunocompromised (Horwitz, M. S., *Adenoviruses*, p. 2149-2171 In B. N. Fields, D. M. Knipe, and P. M. Howley (eds.), *Fields Virology*, Lippincott-Raven Publishers, Philadelphia, PA, 1996). Ad5 is very well understood, can be grown in culture to high titer stocks that are stable, and can replicate in most human cancer cell types (Shenk, T., *Adenoviridae: the viruses and their replication*, p. 2111-2148. In B. N. Fields, D. M. Knipe, and P. M. Howley

(eds.), Fields Virology, Lippincott-Raven, Philadelphia, 1996). Its genome can be manipulated by site-directed mutagenesis and insertion of foreign sequences.

The Ad vectors being investigated for use in anti-cancer and gene therapy are based on recombinant Ad's that are either replication-defective or replication-competent. Typical replication-defective Ad vectors lack the E1A and E1B genes (collectively known as E1) and contain in their place an expression cassette consisting of a promoter and pre-mRNA processing signals which drive expression of a foreign gene. The E1A proteins induce transcription of other Ad genes, and in nontransformed cells they deregulate the cell cycle, induce or repress a variety of cellular genes, and force cells from G₀ into S-phase 48 (White, E., *Semin. Virol.* 8:505-513, 1998; Wold et al., pp. 200-232 *In* A.J. Cann (ed.), DNA Virus Replication: Frontiers in Molecular Biology, Oxford University Press, Oxford). The E1B proteins inhibit cellular apoptosis. *Id.* These vectors are unable to replicate because they lack the E1A genes required to induce Ad gene expression and DNA replication. In addition, the E3 genes are usually deleted because they are not essential for virus replication in cultured cells.

A number of investigators have constructed replication-defective Ad vectors expressing anti-cancer therapeutic proteins. Usually, these vectors have been tested by direct injection of human tumors growing in mouse models. Most commonly, these vectors express the thymidine kinase gene from herpes simplex virus, and the mice are treated with gancyclovir to kill cells transduced by the vector (see e.g., Felzmann et al., *Gene Ther.* 4:1322-1329, 1997). Another suicide gene therapy approach involves injecting tumors with a replication defective Ad vector expressing cytosine deaminase, followed by administration of 5-fluorocytosine (Topf et al., *Gene Ther.* 5:507-513, 1998). Investigators have also prepared and tested replication-defective Ad vectors expressing a cytokine-such as IL-2, IL-12, IL-6, tumor necrosis factor (TNF), type I interferons, or the co-stimulatory molecule B7-1 in the anticipation that the Ad-expressed cytokine will stimulate an immune response, including cytotoxic T-lymphocytes (CTL), against the tumor (Felzmann et al., *supra*; Putzer et al., *Proc. Natl. Acad. Sci. USA* 94:10889-10894, 1997). Other vectors express tumor antigens (e.g. melanoma MART1), proteins that de-regulate the cell cycle and induce apoptosis (p53, pRB, p21^{Kip1/WAF1}, p16^{CDKN2}, and even Ad E1A), and ribozymes. An Ad vector expressing FasL induces apoptosis and tumor regression of a mouse tumor (Arai et al., *Proc. Natl. Acad. Sci. USA* 94:13862-13867, 1997).

Despite these generally positive reports, it is recognized in the art that replication-defective Ad vectors have several characteristics that make them suboptimal for use in therapy. For example, production of replication-defective vectors requires that they be grown on a complementing cell line that provides the E1A proteins in trans. Such cell lines

are fastidious, and generation of virus stocks is time-consuming and expensive. In addition, although many foreign proteins have been expressed from such vectors, the level of expression is low compared to Ad late proteins.

To address these problems, several groups have proposed using replication-competent Ad vectors for therapeutic use. Replication-competent vectors retain Ad genes essential for replication and thus do not require complementing cell lines to replicate. Replication-competent Ad vectors lyse cells as a natural part of the life cycle of the vector. Another advantage of replication-competent Ad vectors occurs when the vector is engineered to encode and express a foreign protein. Such vectors would be expected to greatly amplify synthesis of the encoded protein *in vivo* as the vector replicates. However, in order to prevent RC vectors from damaging normal tissues and causing disseminated viremia, it is important that they have some feature that limits their replication to cancer cells.

Wyeth Laboratories developed replication-competent Ad vectors for vaccination purposes, using vaccine strains of Ad serotypes 4, 7, and 5 (Lubeck et al., *AIDS Res. Hum. Retroviruses* 10:1443-1449, 1994). Foreign genes were inserted into the E3 region (with the E3 genes deleted) or into a site at the right end of the genome. Two foreign genes used were hepatitis B surface antigen and the HIV envelope protein. They obtained good expression in culture, and were able to raise antisera in animal models. Phase I human trials were ambiguous, and the project was mostly abandoned.

Onyx Pharmaceuticals recently reported on adenovirus-based anti-cancer vectors which are replication deficient in non-neoplastic cells but which exhibit a replication phenotype in neoplastic cells lacking functional p53 and/or retinoblastoma (pRB) tumor suppressor proteins (U.S. Patent No. 5,677,178; Heise et al., *Nature Med.* 6:639-645, 1997; Bischoff et al., *Science* 274:373-376, 1996). This phenotype is reportedly accomplished by using recombinant adenoviruses containing a mutation in the E1B region that make the encoded E1B-55K protein incapable of binding to p53 and/or a mutation(s) in the E1A region which make the encoded E1A protein (p289R or p243R) incapable of binding to pRB and/or the cellular 300 kD polypeptide and/or the 107 kD polypeptide. E1B-55K has at least two independent functions: it binds and inactivates the tumor suppressor protein p53, and it is required for efficient transport of Ad mRNA from the nucleus. Because these E1B and E1A viral proteins are involved in forcing cells into S-phase, which is required for replication of adenovirus DNA, and because the p53 and pRB proteins block cell cycle progression, the recombinant adenovirus vectors described by Onyx should replicate in cells defective in p53 and/or pRB, which is the case for many cancer cells, but not in cells with wild-type p53 and/or pRB. Onyx has reported that replication of an adenovirus lacking E1B-55K, which is named ONYX-015, was restricted to p53-minus cancer cell lines (Bischoff et al., *supra*), and

that ONYX-015 slowed the growth or caused regression of a p53-minus human tumor growing in nude mice (Heise et al., *supra*). Others have challenged the Onyx report claiming that replication of ONYX-015 is independent of p53 genotype and occurs efficiently in some primary cultured human cells (Harada and Berk, *J. Virol.* 73:5333-5344, 1999). It is now known that ONYX-015 can replicate in cells with wild-type p53 (Goodrum et al., *J. Virol.* 72:9479-9490, 1998; Harada et al., *J. Virol.* 73:5333-5344, 1999; Hay et al., *Hum. Gene Ther.* 10:579-590, 1999; Rothmann et al., *J. Virol.* 72:9470-9478, 1998; Turnell et al., *J. Virol.* 73:2074-2083, 1999). ONYX-015 does not replicate as well as wild-type adenovirus because E1B-55K is not available to facilitate viral mRNA transport from the nucleus. Also, ONYX-015 expresses less ADP than wild-type virus (see Example 1 below).

As an extension of the ONYX-015 concept, a replication-competent adenovirus vector was designed that has the gene for E1B-55K replaced with the herpes simplex virus thymidine kinase gene (Wilder et al., *Gene Therapy* 6:57-62, 1999). The group that constructed this vector reported that the combination of the vector plus gancyclovir showed a therapeutic effect on a human colon cancer in a nude mouse model (Wilder et al., *Cancer Res.* 59:410-413, 1999). However, this vector lacks the gene for ADP, and accordingly, the vector will lyse cells and spread from cell-to-cell less efficiently than an equivalent vector that expresses ADP. The gene for ADP is also lacking in another replication-competent adenovirus vector that has been described, in which a minimal enhancer/promoter of the human prostate specific antigen was inserted into the adenovirus E1A enhancer/promoter (Rodriguez et al., *Cancer Res.* 57:2559-2563, 1997). Another strategy for replication-competent vector improvement is to place replication under the control of tissue-specific promoters. One group replaced the basal E1A promoter with a modified promoter for α -fetoprotein (AFP) (Hallenbeck et al., *Hum. Gene Ther.* 10:1721-1733, 1999). AFP is expressed in the liver during development, but it is not expressed in adults. However, it is expressed in 70-80% of patients with hepatocellular carcinoma. Growth of this vector was limited to AFP-expressing cells and the vector showed some suppression of xenotransplants. *Id.* Calydon, Inc. also developed adenoviral vectors which overexpress ADP in cells that express either the liver-specific gene AFP or prostate-specific genes, such as prostate-specific antigen (PSA) and probasin (PB). In the Calydon vectors, the E1A or E1B promoter is replaced by either the AFP promoter, PSA promoter, or PB promoter, thereby enabling the ADP expressing adenovirus to replicate in liver tissue and hepatocellular carcinomas or prostate tissue and prostate cancer tissue, respectively ("Adenovirus Vectors Specific for Cells Expressing Alpha-Fetoprotein and Methods of Use Thereof", WO98/39465;

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"Adenovirus Vectors Specific for Cells Expressing Androgen Receptor and Methods of Use Thereof", U.S. Pat. No. 6,197,293).

A series of RC vectors has also been developed that have expression of the E1A and E1B genes dependent on the prostate tumor-specific prostate specific antigen (PSA) and kallikrein promoters/enhancers (Rodriguez et al., *Cancer Res.* 60:1196, 1997; Yu et al., *Cancer Res.* 59:4200-4203, 2000; Yu et al., *Cancer Res.* 59:1498-1504, 1999).

Thus, there is a continuing need for vectors that replicate and spread efficiently in tumors but that can be modified such that they replicate poorly or not at all in normal tissue.

Summary of the Invention

Briefly, therefore, the present invention is directed to novel vectors which are replication competent in neoplastic cells and which overexpress an adenovirus death protein (ADP). The work reported herein demonstrates the discovery that overexpression of ADP by a recombinant adenovirus allows the construction of a replication-competent adenovirus that kills neoplastic cells and spreads from cell-to-cell at a rate similar to or faster than that exhibited by adenoviruses expressing wild-type levels of ADP, even when the recombinant adenovirus contains a mutation that would otherwise reduce its replication rate in non-neoplastic cells. This discovery was unexpected because it could not have been predicted from what was known about adenovirus biology that Ad vectors overexpressing ADP remain viable and that the infected cells are not killed by the higher amounts of ADP before the Ad vector produces new virus particles that can spread to other tumor cells. Indeed, naturally-occurring adenoviruses express ADP in low amounts from the E3 promoter at early stages of infection, and begin to make ADP in large amounts only at 24-30 h p.i., once virions have been assembled in the cell nucleus. It is believed that other non-adenoviral vectors can be used to deliver ADP's cell-killing activity to neoplastic cells, including other viral vectors and plasmid expression vectors.

Thus, in one preferred embodiment, the ADP-expressing vector comprises a recombinant adenovirus lacking expression of at least one E3 protein selected from the group consisting of: gp19K; RID α (also known as 10.4K); RID β (also known as 14.5K) and 14.7K. Because these E3 proteins inhibit immune-mediated inflammation and/or apoptosis of Ad-infected cells, it is believed that a recombinant adenovirus lacking one or more of these E3 proteins will stimulate infiltration of inflammatory and immune cells into a tumor treated with the adenovirus and that this host immune response will aid in destruction of the tumor as well as tumors that have metastasized. The ADP expressed by preferred embodiments comprises a naturally-occurring amino acid sequence from a human adenovirus of subgroup C, namely Ad1, Ad2, Ad5 and Ad6.

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particularly viral vectors that kill the host cell as part of their life cycle. In preferred
embodiments, a recombinant adenovirus has a replication-restricted phenotype because the
recombinant adenovirus is incapable of expressing an E1A viral protein which binds the pRB
and the p300/CBP proteins or because the E4 promoter has been substituted with a promoter
5 that is activated only in neoplastic cells and/or cells of a specific tissue.

In yet another embodiment, the invention provides a vector which overexpresses ADP
and whose replication is under the control of a tissue specific promoter, tumor specific
promoter or an inducible promoter. In preferred embodiments, the vector comprises a
recombinant adenovirus in which the tissue specific promoter or inducible promoter is
10 substituted for the E4 promoter. Such vectors are useful for restricting replication of the
vector and its ADP-mediated cell killing to cells of a particular type or to cells exposed to an
exogenous agent that activates the promoter. A preferred tissue-specific or inducible vector
also expresses a phenotype that restricts its replication to neoplastic cells.

In yet another embodiment, the invention provides a vector which overexpresses ADP
15 but which is not restricted to tumors by a specific genetic modification. Such a vector is more
destructive to neoplastic cells than even the naturally occurring Ad's of subgroup C. In
preferred embodiments, this vector could be used for patients with terminal cancer not
treatable by another method, and who have pre-existing neutralizing antibodies to Ad or to
which neutralizing antibodies can be administered.

20 In still another embodiment, the invention provides a composition comprising a first
recombinant virus which is replication competent in a neoplastic cell and overexpresses the
adenovirus death protein. In one embodiment, the recombinant virus is contained within a
delivery vehicle comprising a targeting moiety that limits delivery of the virus to cells of a
certain type. With this embodiment, the replication-competent vector can be of any ADP-
25 overexpressing configuration described herein. In some embodiments, the composition also
comprises a second recombinant virus which is replication-defective and which expresses an
anti-cancer gene product. In some embodiments, the replication-defective vector may be
engineered to overexpress ADP when replication of this vector is complemented by a
replication-competent vector. The recombinant virus complements spread of the replication-
30 defective virus, as well as its encoded anti-cancer product, throughout a tumor. In preferred
embodiments, the first recombinant virus is a recombinant adenovirus whose replication is
restricted to neoplastic cells and/or which lacks expression of one or more of the E3 gp19K;
RID α ; RID β ; and 14.7K proteins.

In additional embodiments, the invention provides replication-competent vectors that
35 overexpresses an ADP and also expresses an anti-cancer product. As with previous
embodiments, the vector can be of any ADP-overexpressing configuration provided herein.

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Preferably, replication of the virus is engineered to (a) be restricted to neoplastic cells, e.g., by replacing the E4 promoter with a tissue specific or tumor specific promoter and/or (b) lack expression of one or more of the E3 gp19K; RID α ; RID β ; and 14.7K proteins. In some embodiments, the anti-cancer product is inserted into the E3 region.

5 The ADP-expressing vectors and compositions of the invention are useful in a method for promoting death of a neoplastic cell. The method comprises contacting the neoplastic cell with a vector which is replication-competent in the neoplastic cell and which overexpresses ADP. Where the neoplastic cell comprises a tumor in a patient, the vector is administered directly to the tumor or, in other embodiments, the vector is administered to the
10 patient systemically or in a delivery vehicle containing a targeting moiety that directs delivery of the vector to the tumor. In embodiments where the vector is a recombinant virus, the method can also comprise passively immunizing the patient against the virus.

In yet another embodiment of the invention, the vector may be used in combination with radiation therapy. The radiation therapy can be any form of radiation therapy used in the
15 art such as for example, external beam radiation such as x-ray treatment, radiation delivered by insertion of radioactive materials within the body near or at the tumor site such as treatment with gamma ray emitting radionuclides, particle beam therapy which utilizes neutrons or charged particles and the like. In addition, this embodiment encompasses the use of more than one of the vectors of the present invention in a cocktail in combination with
20 radiation therapy.

Another embodiment of the invention involves the use of the recombinant vector in combination with chemotherapy as has been disclosed for other adenovirus vectors (U.S. Patent No. 5,846,945). Chemotherapeutic agents are known in the art and include antimetabolites including pyrimidine-analogue and purine-analogue antimetabolites, plant
25 alkaloids, antitumor antibiotics, alkylating agents and the like. The use of more than one of the vectors of the present invention with a chemotherapeutic agent or agents is also contemplated within this embodiment.

Among the several advantages found to be achieved by the present invention, therefore, may be noted the provision of replication-competent vectors, particularly viruses,
30 which rapidly kill cancer cells and spread from cell-to-cell in a tumor; the provision of such vectors whose replication can be induced or which is restricted to tumors and/or to cells of a certain tissue type; and the provision of compositions and methods for anti-cancer therapy which cause little to no side effects in normal tissues.

Brief Description of the Drawings

35 Figure 1 is a schematic of gene expression in Ad5 (Fig. 1A) and KD3, a preferred embodiment of the invention (Fig. 1B), in which the respective genomes are represented by

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the stippled bars and transcription units represented by arrows above and below the bars, with the E3 proteins listed above the arrows for the E3 transcription unit, and the L1 to L5 families of late mRNA's indicated.

Figure 2 illustrates the overexpression of ADP by KD1, KD3, GZ1, and GZ3 showing an immunoblot of proteins isolated from human A549 cells infected with the indicated viruses and probed with an anti-ADP antibody, with ADP indicating differently glycosylated and proteolytically processed forms of ADP.

Figure 3 illustrates that the E1A *d/1101/1107* mutation referred to in the figure and hereinafter as *d/01/07*, retards expression of late proteins, showing an immunoblot of E1A proteins and late proteins in A549 cells infected with the indicated viruses in the absence (Figs. 3A and 3B) or presence (Figs. 3C and 3D) of *d/327*, which has a wild-type E1A region and has a deletion of all E3 genes but the gene encoding the 12.5K protein (Figs. 3C and 3D). An antiserum specific to the E1A proteins was used for Fig. 3A and 3C. An antiserum raised against Ad5 virions was used for Figs. 3B and 3D.

Figure 4 illustrates that KD1 and KD3 kill cells more efficiently than control viruses that express less or no ADP, showing a graph of the percent of A549 cells infected with the indicated viruses that were viable at the indicated days p.i. as determined by trypan blue exclusion.

Figure 5 is a cell spread assay illustrating that overexpression of ADP enhances spread of virus from cell to cell, showing monolayers infected with the indicated viruses at the indicated PFU/cell which were treated at 7 days p.i. with crystal violet, which stains live cells but not dead cells.

Figure 6 illustrates that KD1 and KD3 replicate well in growing cells but not in growth-arrested cells showing the virus titer extracted from growing or growth arrested HEL-229 cells at various times following infection with 100 PFU/ml of the following viruses: *d/309* (Fig. 6A), *d/01/07* (fig. 6B), KD1 (Fig. 6C) and KD3 (Fig 6D).

Figure 7 illustrates that KD1 and KD3 are defective in killing primary human bronchial epithelial cells showing these cell monolayers infected at 30% confluency with 10 PFU/ml of the indicated viruses and stained at 5 days p.i. with neutral red.

Figure 8 illustrates that KD1 and KD3 reduce the growth rate of human A549 cell tumors growing in nude mice, showing in Fig. 8A a graph of average-fold increase in tumor size plotted against the number of weeks following infection of the tumor with buffer or with 5×10^7 PFU at weekly intervals of or the indicated viruses, and showing in Fig. 8B a similar graph of tumors injected once with 5×10^8 PFU of KD3 or GZ3.

Figure 9 illustrates that KD1 and KD3 reduce the growth rate of human Hep3B cell tumors growing in nude mice, showing a graph of average-fold increase in tumor size plotted

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against the number of weeks following injection of the tumor with buffer or with 5×10^7 PFU of d/309, KD1 or KD3 at twice weekly intervals of the indicated viruses.

Figure 10 illustrates that KD1 and KD3 complement the replication and spread of Ad- β -gal, a replication-defective vector that expresses β -galactosidase, using an infectious center assay showing in Fig. 10A a picture of A549 cell monolayers seeded with A549 cells infected with Ad- β -gal alone or with the indicated viruses, with Figs 10B and 10C showing close-up views of two of the monolayers of Fig. 10A.

Figure 11 is a bar graph illustrating that KD1 and KD3 increase the expression of luciferase in human Hep3B cell tumors growing in nude mice, using an assay in which tumors were injected with the indicated combinations of viruses, then were extracted 2 weeks p.i. and assayed for luciferase activity. The numbers in parentheses indicated the fold increase in luciferase activity compared to that of the Adluc vector plus buffer.

Figure 12 is a graph showing the results of a standard plaque development assay for KD1 and KD1-SPB on A549 cells engineered to express the TTF1 transcription factor (A549/TTF1) and the parental 549 cells, in which data are plotted as the number of plaques observed on a particular day in the assay divided by the final number of plaques observed for that virus multiplied by 100.

Figure 13 is a cell spread assay for KD1 and KD1-SPB on H441 cells and Hep3B cells, where cells were infected with the indicated amounts of KD1 or KD1-SPB and H441 cells and Hep3B cells were stained with crystal violet at 5 days p.i. and 8 days p.i., respectively.

Figure 14 is a graph showing the results of a standard plaque development assay for d/309 and two preferred embodiments of the invention, GZ1 and GZ3, in which data are plotted as the number of plaques observed on a particular day in the assay divided by the final number of plaques observed for that virus multiplied by 100.

Figure 15 is a cell spread assay illustrating that the combination of KD1, KD3, GZ1, or GZ3 with x-ray radiation is more effective in destroying A549 cell monolayers than is virus vector alone or radiation alone, wherein cells were infected with the indicated amounts of the indicated viruses, radiated with 600 centigrays (cGy) of x-radiation (bottom panel), or mock radiated (top panel), then stained with crystal violet at 6 days p.i.

Figure 16 is a graph of a cell spread assay illustrating that 10^{-3} PFU of KD1, KD3, GZ1, or GZ3 used in combination with 150, 300, or 600 centigrays of radiation is more effective in destroying A549 cell monolayers than virus vector alone or radiation alone. Cell viability is based on the amount of crystal violet extracted from the culture wells, using the mock-infected non-radiated well as 100% viability.

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Figure 17 illustrates that the combination of KD3 or GZ3 plus x-ray radiation is more effective in reducing the growth of A549 cell tumors growing in nude mice than KD3 alone or GZ3 alone.

Figure 18 illustrates a structure-function analysis of ADP, showing in Fig. 18A the amino acid sequence of the adenovirus death protein encoded by Ad2, with the various putative domains and glycosylation sites labeled and showing in Fig. 18B a schematic of the ADP gene in *rec700* and in the indicated deletion mutants, with the right column summarizing the death promoting phenotype of the various mutants as a percentage of the wild-type phenotype.

Figures 19A and 19B illustrate a cell viability assay of the indicated ADP mutants showing a graph of viability as determined by trypan blue exclusion plotted against hours (Fig. 19A) or days (Fig. 19B) postinfection.

Figure 20 depicts the amino acid sequence, shown in single letter code, for the ADP proteins of Ad1, Ad2, Ad5, and Ad6 (SEQ ID NOS:5-8), for the Ad2 ADP mutants *dl716*, *dl715*, *dl714*, and *dl737* (SEQ ID NOS:9-12), and for the putative luminal domain (SEQ ID NO:17), the transmembrane domain (SEQ ID NO:18), the cytosolic basic-proline domain (SEQ ID NO:19), and the remainder of the cytosolic domain (SEQ ID NO:20) of the ADP protein of Ad2.

Figure 21 presents the complete nucleotide sequence of the genome of Ad5.

Figure 22 presents the complete nucleotide sequence of the genome of KD1 (SEQ ID NO:1).

Figure 23 presents the complete nucleotide sequence of the genome of KD3 (SEQ ID NO:2).

Figure 24 is a schematic of the following vectors: A. Ad5. The stippled bar indicates the DNA genome of 36 kbp. The open arrow indicates the immediate early E1A transcription unit, and the black arrows are the delayed early E1B, E2, E3, and E4 transcription units. The hatched arrows indicate the five families of major late mRNAs, and also the ADP mRNA, which is synthesized as part of the major late transcription unit. Each major late mRNA has a tripartite leader (leaders 1, 2, and 3) spliced to its 5' terminus. B. dl309. dl309 is identical to Ad5 except it has the E3-RID and E3-14.7K genes deleted. dl309 expresses ADP at levels similar to Ad5. C. KD1. KD1 has two small deletions (indicated by "X" marks) in the E1A gene that abolish binding of the E1A proteins to pRB or p300/CBP. It lacks all E3 genes except adp. ADP is expressed earlier in infection and in greater abundance than is ADP from Ad5 or dl309 Doronin et al., *J. Virol.* 74:6147-6155. D. KD1-SPB. KD1-SPB is identical to KD1, except it has the E4 promoter replaced by the promoter for Surfactant Protein B (SPB-P).

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Figure 25 presents graphs illustrating that KD1-SPB grows as well as KD1 in H441 lung carcinoma cells but much more poorly than KD1 in Hep 3B hepatoma cells. CsCl-banded stocks of KD1-SPB and KD1 were titrated using standard methods (Tollefson et al., p. 1-9 In W.S.M. Wold (ed.), *Adenovirus Methods and Protocols*. Humana Press, Inc., Totowa, NJ, 1998) on 293-E4 or 293 cells (A), or on A549 cells (B). The data are plotted as the number of plaques seen on any day of the plaque assay as a percentage of the number of plaques seen on the final day of the assay (Tollefson et al., *Virology* 220:152-162, 1996).

Figure 26 presents micrographs illustrating that KD1-SPB induces CPE in H441 cells but not Hep 3B cells. H441 and Hep 3B monolayers were mock-infected or infected with 10 PFU/cell of KD1 or KD1-SPB, then photographed under phase contrast at 4 or 7 days p.i.

Figure 27 depicts Southern hybridizations and a graph illustrating that KD1-SPB DNA is synthesized efficiently in H441 but not Hep 3B cells. H441 or Hep 3B cells were infected with 10 PFU/cell of KD1 or KD1-SPB. Total genomic DNA was isolated at 0, 5, 24, 48, 72, and 96 h p.i., digested with HindIII, resolved by agarose gel electrophoresis, blotted, and hybridized with ³²P-labeled Ad DNA. A. Autoradiogram. B. PhosphorImager quantitation of the DNA bands in Panel A.

Figure 28 presents graphs depicting single step growth curves showing that KD1-SPB grows well in H441 but not Hep 3B cells. Cells were infected with 10 PFU/cell of KD1 or KD1-SPB. Vectors were extracted at the indicated days p.i. and titers determined by plaque assay.

Figure 29 depicts immunoblots showing that KD1-SPB expresses E4ORF3 and ADP in H441 but not Hep 3B cells. Cells were infected with 10 PFU/cell of KD1 or KD1-SPB. At 24 h p.i., protein extracts were analyzed for E1A, E4ORF3, and ADP using specific antisera. The E1A proteins appear as multiple bands. ADP appears as two bands; the upper band is glycosylated and the lower band is a proteolytically cleaved species (Scaria et al., *Virology* 191:743-753, 1992; Tollefson et al., *J. Virol.* 66:3633-3642).

Figure 30 depicts immunofluorescence micrographs showing that KD1-SPB expresses E4ORF3 in H441 but not Hep 3B cells. Cells growing on coverslips were infected with 20 PFU/cell of KD1, KD1-SPB, or dl309 (wild-type). At 48 h (Panel A) or 6 days (Panel B), cells were fixed and stained with a rabbit polyclonal antipeptide antiserum against E4ORF3. Photographs were taken using a 100X Planapo lens. Each panel shows about 8 nuclei. This figure is part of the same experiment shown in Figure 31.

Figure 31 depicts immunofluorescence micrographs showing that KD1-SPB does not express E2-DBP or fiber efficiently in Hep 3B cells. Hep 3B cells were infected with 20 PFU/cell of KD1-SPB or KD1. At 48 h (A) or 6 days (B) p.i., cells were fixed and double-stained using a rabbit polyclonal antiserum against DBP and a mouse monoclonal antibody

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against fiber. The same fields are shown for DBP and fiber. This figure is part of the same experiment shown in Figure 30.

Figure 32 presents graphs illustrating that KD1-SPB lyses H441 but not Hep 3B as efficiently as KD1. H441 or Hep 3B cells were mock-infected or infected with 20 PFU/cell of KD1 or KD1-SPB. Cell lysis was determined by release of lactate dehydrogenase from the cells into the medium.

Figure 33 presents graphs illustrating that KD1-SPB suppresses growth of H441 tumors in nude mice equally as well as KD1. Tumor cells were injected into flanks of nude mice and allowed to grow to about 100 μ l (H441) or 150 μ l (Hep 3B) volumes. Tumors (n = 10) were injected with DMEM (mock) or with 5×10^7 PFU of KD1 or KD1-SPB. Injections of the viruses were repeated twice weekly for 3 weeks to a total dose of 3.0×10^8 PFU per tumor. Tumors were measured and the mean fold-increase in tumor size was calculated.

Description of the Preferred Embodiments

In accordance with the present invention, it has been discovered that overexpression of ADP by a recombinant adenovirus results in faster lysis of cells and spread of the virus throughout a cell monolayer than viruses expressing wild-type levels of ADP. It has also been discovered that this function for ADP is manifest in an adenovirus that contains E1A mutations that restrict adenoviral replication to neoplastic cells. Thus, vectors which are both replication competent in neoplastic cells and which overexpress ADP should be useful in anti-cancer therapy.

In the context of this disclosure, the following terms will be defined as follows unless otherwise indicated:

"Naturally-occurring" as applied to an object such as a polynucleotide, polypeptide, or virus means that the object can be isolated from a source in nature and has not been intentionally modified by a human.

"Neoplastic cell" means a cell which exhibits an aberrant growth phenotype characterized by a significant loss of control of cell proliferation and includes actively replicating cells as well as cells in a temporary non-replicative resting state (G_1 or G_2). A neoplastic cell may have a well-differentiated phenotype or a poorly-differentiated phenotype and may comprise a benign neoplasm or a malignant neoplasm.

"Recombinant virus" means any viral genome or virion that is different than a wild-type virus due to a deletion, insertion, or substitution of one or more nucleotides in the wild-type viral genome. The recombinant virus can have changes in the number of amino acid sequences encoded and expressed or in the amount or activity of proteins expressed by the virus. In particular, the term includes recombinant viruses generated by the intervention of a human.

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"Replication-competent" as applied to a vector means that the vector is capable of replicating in normal and/or neoplastic cells. As applied to a recombinant virus, "replication-competent" means that the virus exhibits the following phenotypic characteristics in normal and/or neoplastic cells: cell infection; replication of the viral genome; and production and release of new virus particles; although one or more of these characteristics need not occur at the same rate as they occur in the same cell type infected by a wild-type virus, and may occur at a faster or slower rate. Where the recombinant virus is derived from a virus such as adenovirus that lyses the cell as part of its life cycle, it is preferred that at least 5 to 25% of the cells in a cell culture monolayer are dead 5 days after infection. Preferably, a replication-competent virus infects and lyses at least 25 to 50%, more preferably at least 75%, and most preferably at least 90% of the cells of the monolayer by 5 days post infection (p.i.).

"Replication-defective" as applied to a recombinant virus means the virus is incapable of, or is greatly compromised in, replicating its genome in any cell type in the absence of a complementing replication-competent virus. Exceptions to this are cell lines such as 293 cells that have been engineered to express adenovirus E1A and E1B proteins.

"Replication-restricted" as applied to a vector of the invention means the vector replicates better in a dividing cell, i.e. either a neoplastic cell or a non-neoplastic, dividing cell, than in a cell of the same type that is not neoplastic and/or not dividing, which is also referenced herein as a normal, non-dividing cell. Preferably, a replication-restricted virus kills at least 10% more neoplastic cells than normal, non-dividing cells in cell culture monolayers of the same size, as measured by the number of cells showing cytopathic effects (CPE) at 5 days p.i. More preferably, between 25% and 50%, and even more preferably, between 50% and 75% more neoplastic than normal cells are killed by a replication-restricted virus. Most preferably, a replication-restricted adenovirus kills between 75% and 100% more neoplastic than normal cells in equal sized monolayers by 5 days p.i.

In one embodiment the invention provides a vector that is replication-competent in neoplastic cells and which overexpresses an ADP. Vectors useful in the invention include but are not limited to plasmid-expression vectors, bacterial vectors such as *Salmonella* species that are able to invade and survive in a number of different cell types, vectors derived from DNA viruses such as human and non-human adenoviruses, adenovirus associated viruses (AAVs), poxviruses, herpesviruses, and vectors derived from RNA viruses such as retroviruses and alphaviruses. Preferred vectors include recombinant viruses engineered to overexpress an ADP. Recombinant adenoviruses are particularly preferred for use as the vector, especially vectors derived from Ad1, Ad2, Ad5 or Ad6.

Vectors according to the invention overexpress ADP. As applied to recombinant Ad and AAV vectors, the term "overexpresses ADP" means that more ADP molecules are made

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per viral genome present in a dividing cell infected by the vector than expressed by any previously known recombinant adenoviral vector or AAV in a dividing cell of the same type. As applied to other, non-adenoviral vectors, "overexpresses ADP" means that the virus expresses sufficient ADP to lyse a cell containing the vector.

- 5 Vectors overexpressing ADP can be prepared using routine methodology. See, e.g., *A Laboratory Cloning Manual*, 2nd Ed., vol. 3, Sambrook et al., eds., Cold Spring Harbor Laboratory Press, 1989. For example, a polynucleotide encoding the ADP can be cloned into a plasmid expression vector known to efficiently express heterologous proteins in mammalian cells. The polynucleotide should also include appropriate termination and polyadenylation
10 signals. Enhancer elements may also be added to the plasmid to increase the amount of ADP expression. Viral vectors overexpressing ADP can be prepared using similar materials and techniques.

- Where the virus is a recombinant adenovirus, overexpression of ADP can be achieved in a multitude of ways. In general, any type of deletion in the E3 region that removes a splice
15 site for any of the E3 mRNAs will lead to overexpression of the mRNA for ADP, inasmuch as more of the E3 pre-mRNA molecules will be processed into the mRNA for ADP. This is exemplified in the KD1, KD3, GZ1 and GZ3 vectors (SEQ ID NOS:1-4) whose construction is described below. Other means of achieving overexpression of ADP in Ad vectors include, but are not limited to: insertion of pre-mRNA splicing and cleavage/polyadenylation signals
20 at sites flanking the gene for ADP; expression of ADP from another promoter, e.g. the human cytomegalovirus promoter, inserted into a variety of sites in the Ad genome; and insertion of the gene for ADP behind the gene for another Ad mRNA, together with a sequence on the 5' side of the ADP sequence that allows for internal initiation of translation of ADP, e.g. the Ad tripartite leader or a viral internal ribosome initiation sequence.

- 25 The ADP expressed by a vector according to the invention is any polypeptide comprising a naturally-occurring full-length ADP amino acid sequence or variant thereof that confers upon a vector expressing the ADP the ability to lyse a cell containing the vector such that replicated copies of the vector are released from the infected cell. A preferred full-length ADP comprises the ADP amino acid sequence encoded by Ad1, Ad2, Ad5 or Ad6. These
30 naturally-occurring ADP sequences are set forth in SEQ ID NOS:5-8, respectively. ADP variants include fragments and deletion mutants of naturally-occurring adenovirus death proteins, as well as full-length molecules, fragments and deletion mutants containing conservative amino acid substitutions, provided that such variants retain the ability, when expressed by a vector inside a cell, to lyse the cell.

- 35 Conservative amino acid substitutions refer to the interchangeability of residues having similar side chains. Conservatively substituted amino acids can be grouped according

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to the chemical properties of their side chains. For example, one grouping of amino acids includes those amino acids having neutral and hydrophobic side chains (A, V, L, I, P, W, F, and M); another grouping is those amino acids having neutral and polar side chains (G, S, T, Y, C, N, and Q); another grouping is those amino acids having basic side chains (K, R, and H); another grouping is those amino acids having acidic side chains (D and E); another grouping is those amino acids having aliphatic side chains (G, A, V, L, and I); another grouping is those amino acids having aliphatic-hydroxyl side chains (S and T); another grouping is those amino acids having amine-containing side chains (N, Q, K, R, and H); another grouping is those amino acids having aromatic side chains (F, Y, and W); and another grouping is those amino acids having sulfur-containing side chains (C and M). Preferred conservative amino acid substitutions groups are: R-K; E-D, Y-F, L-M; V-I, and Q-H.

As used herein, an ADP variant can also include modifications of a naturally-occurring ADP in which one or more amino acids have been inserted, deleted or replaced with a different amino acid or a modified or unusual amino acid, as well as modifications such as glycosylation or phosphorylation of one or more amino acids so long as the ADP variant containing the modified sequence retains cell lysing activity.

As described below, the inventors herein performed a structure-function analysis of ADP that defined specific domains in ADP required to promote cell death. Using this information, when combined with known recombinant DNA and cloning methodology, it is believed the skilled artisan can readily construct ADP variants of a naturally-occurring adenovirus death protein and test them for cell lysing activity. A preferred ADP deletion mutant comprises an ADP amino acid sequence from any of the deletion mutants *d1716*, *d1715*, *d1714* and *d1737*, whose ADP sequences are set forth in SEQ ID NOS:9-12, respectively).

Where the vector is derived from a virus, it is preferred that the virus lack expression of one or more viral proteins involved in avoiding host anti-viral defenses such as immune-mediated inflammation and/or apoptosis of infected cells. For example, adenovirus contains a cassette of genes that prevents killing of Ad-infected cells by the immune system (Wold et al., *Semin. Virol.*, 1998 (8):515-523, 1998). The E3-14.7K protein and the E3 RID (Receptor Internalization and Degradation) protein, which is a complex consisting of RID α and RID β , inhibit apoptosis of Ad-infected cells induced by tumor necrosis factor (TNF) and the Fas ligand which are expressed on, or secreted by, activated macrophages, natural killer (NK) cells, and cytotoxic lymphocytes (CTLs) (Tollefson et al., *Nature* 392:727-730, 1998). The E3-gp19K protein inhibits CTL-killing of infected cells by blocking transport of MHC class I antigens to the cell surface (Wold et al., *supra*). Thus, it is believed that infection of tumor cells by such viral vectors will stimulate infiltration of inflammatory cells and lymphocytes

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into the tumor, and will not prevent infected tumor cells from apoptosis induced by cytolytic cells of the immune system, or against apoptosis inducing cytokines. For example, it is known that when mice are infected with Ad mutants lacking the E3 gp19K, RID and 14.7K proteins there is a dramatic increase (as compared to E3-positive Ad) in infiltration of inflammatory cells and lymphocytes into the infected tissue (Sparer et al., *J. Virol.* 70:2431-2439, 1996). A similar infiltration of tumors infected by an ADP-expressing viral vector of the invention would be expected to further promote destruction of the tumor by adding an immune system attack to the ADP-mediated killing activity. For example, it is believed that the viral infection will stimulate formation of tumor-specific CTL's that can kill neoplastic cells not only in the tumor but also ones that have metastasized. In addition, it is also expected that vector-specific CTL's will be generated which could attack vector-infected cells if the vector spreads away from the tumor into normal cells. Because viral vectors overexpressing ADP will spread rapidly through the tumor, it is believed these immune mechanisms will have little effect on spread of the vector.

Where the vector is a recombinant adenovirus, it is preferred that the adenovirus lack expression of each of the E3 gp19K, RID, and 14.7K proteins. By "lack expression" and "lacking expression" of a protein(s), it is meant that the viral genome contains one or more mutations that inactivates expression of a functional protein, i.e., one having all the functions of the wild-type protein. The inactivating mutation includes but is not limited to substitution or deletion of one or more nucleotides in the encoding gene(s) that prevents expression of functional transcripts or that results in transcripts encoding nonfunctional translation products. A particularly preferred way to inactivate expression of the Ad E3 gp19K, RID, and 14.7K proteins is by deleting the E3 region containing the genes encoding these proteins. Preferably, one or both of the E3 genes encoding the E3 6.7K and 12.5K proteins are also deleted because, as discussed in the Examples below, it is believed that deletion of most or all of the E3 genes other than the ADP gene facilitates overexpression of ADP mRNA by reducing competition for splicing of the major late pre-mRNAs. Preferred Ad vectors containing an E3 deletion that overexpress ADP are GZ1 (SEQ ID NO:3) and GZ3 (SEQ ID NO:4), whose construction and properties are described in the Examples below.

The invention also provides ADP-expressing vectors whose replication is restricted to dividing cells. Any means known to provide such a replication-restricted phenotype may be used. For example, WO 96/40238 describes microbes that preferentially invade tumor cells as well as methods for identifying and isolating bacterial promoters that are selectively activated in tumors. It is also contemplated that expression of one or more vector proteins essential for replication can be placed under the control of the promoter for a cellular gene whose expression is known to be upregulated in neoplastic cells. Examples of such genes

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include but are not limited to: the breast cancer markers mammaglobin (Watson et al., *Oncogene* 16:817-824, 1998); BRCA1 (Norris et al., *J. Biol. Chem.* 270:22777-22782, 1995) *her2/neu* (Scott et al., *J. Biol. Chem.* 269:19848-19858, 1994); prostate specific antigen (U.S. Patent 5,698,443); surfactant protein B for lung alveoli (Yan et al., *J. Biol. Chem.* 270:24852-24857, 1995); factor VII for liver (Greenberg et al., *Proc. Natl. Acad. Sci. USA* 92:12347-12351, 1995); and survivin for cancer in general (Li et al., *Nature* 396:580-584). Where the vector is an adenovirus, it is contemplated that such tumor-specific promoters can be substituted for the E4 promoter. Because E4 gene products are essential for Ad replication, placing their expression under the control of a tumor-specific promoter should restrict replication of the vector to tumor cells in which the promoter is activated.

Another strategy for restricting replication of ADP-expressing Ad vectors to neoplastic cells is exemplified by the KD1 (SEQ ID NO:1), KD2 (SEQ ID NO:13) and KD3 (SEQ ID NO:2) vectors, whose construction and properties are described in the Examples below. This strategy exploits a pre-existing Ad5 mutant in the E1A gene, named *d1101/1107* (Howe et al., *Proc. Natl. Acad. Sci.*, 87:5883-5887, 1990), also referred to herein as *d101/07*, and which can only grow well in cancer cells. The role of E1A is to drive cells from the G₀ and G₁ phases of the cell cycle into S-phase. This is achieved by two mechanisms, one involving pRB (and family members), and the other involving p300 and the related protein CBP (DePinho, R.A., *Nature* 391:533-536, 1998). One domain in E1A binds members of the pRB family. pRB normally exists in the cell as a complex with the transcription factor E2F-1 and E2F family members (E2F), tethered via E2F to E2F binding sites in promoters of cells expressed in S-phase. Here, pRB acts as a transcriptional co-repressor. E1A binding to pRB relieves this repression, and causes the release of E2F from pRB/E2F complexes. Free E2F then activates promoters of genes expressed in S-phase, e.g. thymidine kinase, ribonucleotide reductase, etc. Another domain in E1A binds the p300/CBP transcription adaptor protein complex. p300/CBP is a transcriptional co-activator that binds many different transcription factors and accordingly is targeted to promoters. p300/CBP has intrinsic histone acetyltransferase activity. E1A binding to p300/CBP is believed to inhibit this histone acetyltransferase activity, allowing acetylation of histones and repression of transcription (Chakravarti et al., *Cell* 96:393-403, 1999; Hamamori et al., *Cell* 96:405-413, 1999). Conceivably, some of the genes that are repressed as a result of E1A interacting with p300/CBP to play a role in blocking the cell cycle, although this is not known. Cancer cells are cycling, so they have free E2F and presumably some p300/CBP-regulated genes are repressed. Consistent with these ideas, E1A must bind both p300/CBP and the pRB family in order to transform primary cells to a constitutively cycling state (Howe et al., *supra*). The mutant *d101/07* lacks both the p300/CBP- and pRB-binding domains and, as expected, it

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replicates very poorly in non-dividing "normal" cells or serum-starved cancer cells, but well in growing cancer cells. As described below, the growth of the KD1 and KD3 vectors, which contain the d/01/07 E1A mutation, is very much better in dividing cancer cells as compared to non-dividing cells. Because the d/01/07 mutant is completely defective in oncogenic transformation of rat cells (Howe et al., *supra*), vectors according to the invention that contain this E1A mutation cannot induce cancer in humans (remote as that may be) through an E1A-dependent mechanism.

The invention also includes vectors overexpressing ADP whose replication is restricted to specific tissues by placing expression of one or more proteins essential for replication under the control of a tissue specific promoter and/or a tumor specific promoter. A number of tissue-specific and/or tumor specific promoters have been described in the art. Non-limiting examples include the surfactant protein B promoter, which is only active in cells containing the TTF1 transcription factor (i.e., type II alveolar cells (Yan et al., *supra*)), as described in U.S. Patent 5,466,596 to Breitman et al., which directs gene expression specifically in cells of endothelial lineage; prostate specific antigen which is expressed in prostate cells (Rodriguez et al., *supra*); human telomerase protein (hTERT) promoter (see, e.g., U.S. Patent No. 6,054,575); and human alpha-lactalbumin gene which is expressed in breast cancer cells (Anderson et al., *Gene Therapy* 6:854-864, 1999). Many other tissue-specific, tumor specific, or tissue-preferred enhancer/promoters have been reported (Miller and Whelan, *Human Gene Therapy* 8:803-815, 1997). As exemplified with the surfactant protein B promoter in Examples 6 and 10, vectors expressing tissue-specific promoters would be expected to show tissue specificity in viral replication, viral spreading, cell lysis, and tumor suppression.

Replication of vectors according to the invention can also be controlled by placing one or more genes essential for vector replication under the control of a promoter that is activated by an exogenous inducing agent, such as metals, hormones, antibiotics, and temperature changes. Examples of such inducible promoters include but are not limited to metallothionein promoters, the glucocorticoid promoter, the tetracycline response promoter, and heat shock protein (hsp) promoters such as the hsp 65 and 70 promoters.

The invention also provides compositions comprising a recombinant vector that overexpresses ADP in an amount effective for promoting death of neoplastic cells and a method comprising administering a therapeutically effective amount of the vector to a neoplastic cell in a patient. It is believed the compositions and methods of the present invention are useful for killing neoplastic cells of any origin and include neoplastic cells comprising tumors as well as metastatic neoplastic cells.

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It is also contemplated that ADP-expressing viral vectors can be administered to neoplastic cells along with a replication-defective virus that expresses an anti-cancer gene product. For example, many replication-defective E1⁻ Ad vectors for use in cancer therapy are well characterized. A limitation of replication-defective vectors is that they only
5 synthesize the therapeutic protein in the cell they initially infect, they cannot spread to other cells. Also, since the genome does not replicate, transcription can only occur from the input genomes, and this could be as low as one copy per cell. In contrast, the genome of replication-competent Ad vectors are amplified by about 10^4 in the cell that was initially infected, providing more templates for transcription. More amplification is achieved as the
10 vector spreads to other cells. By combining replication-defective viral vectors expressing an anti-cancer gene product with replication-competent viral vectors described herein, it is expected that the result will be template amplification and rapid spread of both vectors to surrounding cells. For example, with Ad-based vectors, the burst size for each vector should be large, $\sim 10^4$ PFU/cell, so the probability of co-infection of surrounding cells by both vectors
15 will be high. Thus, both the replication-competent and replication-defective vectors should spread simultaneously through the tumor, providing even more effective anti-cancer therapy.

As an alternative method of delivering an anti-cancer gene product with an ADP overexpressing Ad vector, the anti-cancer gene can be engineered into any of the ADP overexpressing replication-competent vectors described herein, in order to provide both the
20 ADP and the anti-cancer function in a single vector. The anti-cancer gene can be engineered into any appropriate location of the vector, as can be easily determined by the skilled artisan. For example, the anti-cancer gene can be engineered into the E3 region.

Expression of the anti-cancer gene product encoded by the replication-defective vector can be under the control of either constitutive, inducible or cell-type specific
25 promoters. The anti-cancer gene product can be any substance that promotes death of a neoplastic cell. The term "gene product" as used herein refers to any biological product or products produced as a result of the biochemical reactions that occur under the control of a gene. The gene product can be, for example, an RNA molecule, a peptide, a protein, or a product produced under the control of an enzyme or other molecule that is the initial product
30 of the gene, i.e., a metabolic product. For example, a gene can first control the synthesis of an RNA molecule which is translated by the action of ribosomes into a prodrug converting enzyme which converts a nontoxic prodrug administered to a cancer patient to a cell-killing agent; the RNA molecule, enzyme, and the cell-killing agent generated by the enzyme are all gene products as the term is used here. Examples of anti-cancer gene products include but are
35 not limited to cell-killing agents such as apoptosis-promoting agents and toxins; prodrug converting enzymes; angiogenesis inhibitors; and immunoregulatory molecules and antigens

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capable of stimulating an immune response, humoral and/or cellular, against the neoplastic cell.

Apoptosis-promoting agents include but are not limited to the pro-apoptotic members of the BCL-2 family such as BAX, BAD, BID and BIK, as well as antisense molecules which block expression of anti-apoptotic members of the BCL-2 family. Examples of immunoregulatory molecules are cytokines such as tumor necrosis factor, Fas/Apo1/CD95 ligand, tumor necrosis factor related apoptosis inducing ligand, interleukins, macrophage activating factor and interferon γ . Angiogenesis inhibitors include but are not limited to endostatin and angiostatin. Toxins include but are not limited to tumor necrosis factor, lymphotoxin, the plant toxin ricin, which is not toxic to humans due to the lack of ricin receptors in animal cells, and the toxic subunit of bacterial toxins. Examples of pro-drug converting enzymes and pro-drug combinations are described in WO 96/40238 and include thymidine kinase and acyclovir or gancyclovir, and bacterial cytosine deaminase and 5-fluorocytosine.

The therapeutic or pharmaceutical compositions of the present invention can be administered by any suitable route known in the art including for example by direct injection into a tumor or by other injection routes such as intravenous, subcutaneous, intramuscular, transdermal, intrathecal and intracerebral. Administration can be either rapid as by injection or over a period of time as by slow infusion or administration of slow release formulation. For treating tissues in the central nervous system, administration can be by injection or infusion into the cerebrospinal fluid (CSF). When it is intended that a recombinant vector of the invention be administered to cells in the central nervous system, administration can be with one or more agents capable of promoting penetration of the vector across the blood-brain barrier. Preferably, vectors of the invention are administered with a carrier such as liposomes or polymers containing a targeting moiety to limit delivery of the vector to targeted cells. Examples of targeting moieties include but are not limited to antibodies, ligands or receptors to specific cell surface molecules.

Compositions according to the invention can be employed in the form of pharmaceutical preparations. Such preparations are made in a manner well known in the pharmaceutical art. One preferred preparation utilizes a vehicle of physiological saline solution, but it is contemplated that other pharmaceutically acceptable carriers such as physiological concentrations of other non-toxic salts, five percent aqueous glucose solution, sterile water or the like may also be used. It may also be desirable that a suitable buffer be present in the composition. Such solutions can, if desired, be lyophilized and stored in a sterile ampoule ready for reconstitution by the addition of sterile water for ready injection. The primary solvent can be aqueous or alternatively non-aqueous.

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The carrier can also contain other pharmaceutically-acceptable excipients for modifying or maintaining the pH, osmolality, viscosity, clarity, color, sterility, stability, rate of dissolution, or odor of the formulation. Similarly, the carrier may contain still other pharmaceutically-acceptable excipients for modifying or maintaining release or absorption or penetration across the blood-brain barrier. Such excipients are those substances usually and customarily employed to formulate dosages for parenteral administration in either unit dosage or multi-dose form or for direct infusion into the cerebrospinal fluid by continuous or periodic infusion.

It is also contemplated that certain formulations containing ADP-expressing vectors are to be administered orally. Such formulations are preferably encapsulated and formulated with suitable carriers in solid dosage forms. Some examples of suitable carriers, excipients, and diluents include lactose, dextrose, sucrose, sorbitol, mannitol, starches, gum acacia, calcium phosphate, alginates, calcium silicate, microcrystalline cellulose, polyvinylpyrrolidone, cellulose, gelatin, syrup, methyl cellulose, methyl- and propylhydroxybenzoates, talc, magnesium stearate, water, mineral oil, and the like. The formulations can additionally include lubricating agents, wetting agents, emulsifying and suspending agents, preserving agents, sweetening agents or flavoring agents. The compositions may be formulated so as to provide rapid, sustained, or delayed release of the active ingredients after administration to the patient by employing procedures well known in the art. The formulations can also contain substances that diminish proteolytic degradation and promote absorption such as, for example, surface active agents.

The specific dose is calculated according to the approximate body weight or body surface area of the patient or the volume of body space to be occupied. The dose will also be calculated dependent upon the particular route of administration selected. Further refinement of the calculations necessary to determine the appropriate dosage for treatment is routinely made by those of ordinary skill in the art. Such calculations can be made without undue experimentation by one skilled in the art. Exact dosages are determined in conjunction with standard dose-response studies. It will be understood that the amount of the composition actually administered will be determined by a practitioner, in the light of the relevant circumstances including the condition or conditions to be treated, the choice of composition to be administered, the age, weight, and response of the individual patient, the severity of the patient's symptoms, and the chosen route of administration. Dose administration can be repeated depending upon the pharmacokinetic parameters of the dosage formulation and the route of administration used.

The invention also contemplates passively immunizing patients who have been treated with a viral vector overexpressing ADP. Passive immunization can include

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administering to the patient antiserum raised against the viral vector, or gamma-globulin or vector-specific purified polyclonal or monoclonal antibodies isolated from the antiserum. Preferably, the patient is passively immunized after a time period sufficient for the viral vector to replicate in and spread through the tumor.

- 5 Preferred embodiments of the invention are described in the following examples. Other embodiments within the scope of the claims herein will be apparent to one skilled in the art from consideration of the specification or practice of the invention as disclosed herein. It is intended that the specification, together with the examples, be considered exemplary only, with the scope and spirit of the invention being indicated by the claims which follow the
- 10 examples.

Example 1

This example illustrates the construction and characterization of the KD1 and KD3 anti-cancer vectors.

- To construct KD1, the inventors deleted the entire E3 region of a unique plasmid, leaving behind only a unique *PacI* site for cloning. The starting plasmid was pCRII, purchased from Invitrogen, containing the Ad5 *Bam*HIA fragment having a deletion of all the E3 genes; the E3 deletion is identical to that for KD1 and GZ3, the sequences of which are given in SEQ ID NO:1 and SEQ ID NO:4, respectively. The ADP gene from Ad5 was cloned into the *PacI* site, then built into the E3 region of the genome of the Ad5 E1A mutant named *dl01/07*. This was done by co-transfecting into human embryonic kidney 293 cells the
- 15 aforementioned *Bam*HIA fragment containing the ADP gene together with the overlapping *Eco*RIA restriction fragment obtained from *dl01/07*. Complete viral genomes are formed within the cell by overlap recombination between the Ad sequences in the *Bam*HIA fragment in the plasmid and the *Eco*RIA fragment. KD3 was constructed in the same way except the E3 gene for the 12.5K protein was retained in the starting plasmid. A vector named KD2, which marginally overexpress ADP, was also prepared. Plaques of each recombinant Ad were picked, screened, purified, expanded into CsCl-banded stocks, sequenced, titered, and characterized. GZ1 and GZ3 are Ad vectors that are identical to KD1 and KD3, respectively, except that GZ1 and GZ3 have wild-type E1A sequences as found in Ad5 or in the Ad5 mutant *dl309*. GZ1 and GZ3 were constructed as described for KD1 and KD3 except that the
- 20 *Eco*RIA fragment of Ad5 was used for GZ1 and GZ3.

- KD1 and KD3 were characterized in cell culture by infecting the human A549 lung carcinoma cell line with high titer ($1-8 \times 10^{10}$ plaque forming units [PFU] per ml) virus stocks of one of these recombinant vectors, or with one of the control viruses *dl01/07*, *dl309*, *dl327*, and Ad5 (wt). Fifty PFU per cell were used for each virus. The descriptions of these viruses as well as some other viruses used in these examples are presented in Table 1.
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Table 1: Description of mutations in viruses:

us	RNA				REGION	
	E1	VA	E3	E4		
101/1107	<i>dl</i> 1101: deletion of Ad5 bp 569-634 <i>dl</i> 1107: deletion of Ad5 bp 890-928	From <i>dl</i> 309 deletion of Ad5 bp 10594-10595	From <i>dl</i> 309 deletion of Ad5 bp 28597-28602; deletion-substitution Ad5 bp 3005-30750, insert 642 bp DNA of unknown origin	wild type		
11	<i>dl</i> 1101: deletion of Ad5 bp 569-634 <i>dl</i> 1107: deletion of Ad5 bp 890-928	From <i>dl</i> 309 deletion of Ad5 bp 10594-10595	deletion of Ad5 bp 27858-2760, TAA inserted; deletion of Ad5 bp 27982-28134; deletion of Ad5 bp 28395-29397, insert CCTTAATTAAA; deletion of Ad5 bp 29783-30883, insert TTAATTAAAGG	wild type		
12	<i>dl</i> 1101: deletion of Ad5 bp 569-634 <i>dl</i> 1107: deletion of Ad5 bp 890-928	From <i>dl</i> 309 deletion of Ad5 bp 10594-10595	<i>dl</i> 309 background, gp19K mutated deletion of Ad5 bp 28597-28602; deletion-substitution Ad5 bp 3005-30750, insert 642 bp DNA of unknown origin; deletion of Ad5 bp 28788-28789, insert TTAATTAA	wild type		
13	<i>dl</i> 1101: deletion of Ad5 bp 569-634 <i>dl</i> 1107: deletion of Ad5 bp 890-928	From <i>dl</i> 309 deletion of Ad5 bp 10594-10595	deletion of Ad5 bp 28598-29397, deletion of Ad5 bp 29783-30469	wild type		
14	wt	wild type	deletion of Ad5 bp 27858-2760, TAA inserted; deletion of Ad5 bp 27982-28134; deletion of Ad5 bp 28395-29397, insert CCTTAATTAAA; deletion of Ad5 bp 29783-30883, insert TTAATTAAAGG	wild type		

	wild type	wild type	wild type	wild type
01/1107-	<i>d11101</i> : deletion of Ad5 bp 569-634 <i>d11107</i> : deletion of Ad5 bp 890-928	From <i>d1309</i> deletion of Ad5 bp 10594-10595	deletion of Ad5 bp 28598-29397; deletion of Ad5 bp 29783-30469 From <i>d1309</i> deletion of Ad5 bp 28597-28602; deletion-substitution Ad5 bp 3005-30750, insert 642 bp DNA of unknown origin	E4 promoter deletion-substitution: deletion of Ad5 bp 35623-35775, insert SP-B 500 promoter flanked by BstI 1071 sites
1-SPB	<i>d11101</i> : deletion of Ad5 bp 569-634 <i>d11107</i> : deletion of Ad5 bp 890-928	From <i>d1309</i> deletion of Ad5 bp 10594-10595	deletion of Ad5 bp 27848-2760, TAA inserted; deletion of Ad5 bp 27982-28134; deletion of Ad5 bp 28395-29397, insert CCTTAAATTAAA; deletion of Ad5 bp 29783-30883, insert TTAAATTAAGG	E4 promoter deletion-substitution: deletion of Ad5 bp 35623-35775, insert SP-B 500 promoter flanked by BstI 1071 sites
1-SPB	<i>d11101</i> : deletion of Ad5 bp 569-634 <i>d11107</i> : deletion of Ad5 bp 890-928	From <i>d1309</i> deletion of Ad5 bp 10594-10595	deletion of Ad5 bp 28598-29397; deletion of Ad5 bp 29783-30469	E4 promoter deletion-substitution: deletion of Ad5 bp 35623-35775, insert SP-B 500 promoter flanked by BstI 1071 sites

Using a polymerase chain reaction (PCR)-based protocol, an in-frame stop codon was introduced into the gene for the E3-gp19K protein in the E3 region of the Ad5 mutant *dI309* (Jones and Shenk, *Cell* 17:683-689, 1979). The mutagenesis was conducted using a *SmaI*-*Bst*1107I fragment, nucleotides 28,390 to 29,012 in the Ad5 genome, which was then substituted for the equivalent fragment in *dI309*. *dI01/07* is the parent for KD1 and KD3. In turn, the Ad5 mutant named *dI309* is the parent of *dI01/07*, i.e. *dI309* is identical to *dI01/07* except that *dI309* does not have the E1A mutation. Both *dI01/07* and *dI309* have deletions of the genes for the E3 RID α , RID β and 14.7K proteins but retain the gene for ADP. The Ad5 mutant *dI327* has wild-type E1A, it lacks the gene for ADP, and it lacks all other E3 genes except the one for the 12.5K protein.

At 24 and 36 hours post-infection (h p.i.), proteins were extracted from the A549 cells and analyzed for ADP by immunoblot using a rabbit antiserum against ADP (Tollefson et al., *J. Virol.* 66:3633-3642, 1992). The results are shown in Figure 2. Much more ADP was detected at 24 and 36 h p.i. in KD1- and KD3-infected cells than in cells infected with *dI01/07*. Also, much more ADP was synthesized by GZ1 and GZ3 than *dI309* or the other viruses. Most importantly, KD1, KD3, GZ1, and GZ3 expressed much more ADP at 24 h p.i. than did *dI01/07* or *dI309* (Fig. 2). This result is consistent with an observation discussed below that the cells infected with KD1, KD3, GZ1, or GZ3 lyse faster, and that these viruses spread from cell to cell faster than *dI01/07* or *dI309*. It is noteworthy that KD1, KD3, GZ1, and GZ3 express much more ADP at 24 and 36 h p.i. than the Ad5 mutant *dI1520* (Fig. 2); *dI1520* is the original name given to ONYX-015 (Heise et al., *Nature Medicine* 3:639-645, 1997). As expected, no ADP was detected in cells infected with *pm734.1* (Fig. 2), a mutant that lacks amino acids 1 to 48 in ADP (Tollefson et al., *J. Virol.* 70:2296-2306, 1996). Expression of the E1A proteins by *dI01/07*, KD1, KD2, and KD3 was slightly less than by Ad5, *dI309*, or *dI327*, and as expected from the *dI01/07* deletion, the proteins were smaller (Fig. 3A). *dI327* is isogenic with *dI324* (Thimmappaya et al., 1982 *Cell* 31:543-51, 1983), and it lacks the gene for ADP and all other E3 proteins except the 12.5K protein.

The amount of ADP detected in the KD1 and KD3 infected cells is significantly higher than the amount detected in the *dI309* infected cells (Fig. 2). If one takes into consideration the fact that the viruses with the E1A mutation replicate somewhat slower, as evidenced in by the delayed appearance of the late proteins (Fig. 3B), it is clear that KD1 and KD3 express much more ADP per viral genome present in the cell than *dI309*. This finding is supported by the fact that when A549 cells are coinfecting with a virus containing the E1A mutation and *dI327*, which lacks ADP but has wild-type E1A, the replication rates of the E1A mutant viruses speed up, as indicated by earlier appearance of late proteins (compare Figs. 3B

and 3D). Thus, *dI327* complements the E1A mutation. In conclusion, these experiments demonstrate that ADP is dramatically overexpressed by KD1, KD3, GZ1, and GZ3. ADP is marginally overexpressed by KD2 (not shown).

Example 2

5 This example illustrates that KD1 and KD3 lyse cells more rapidly and spread from cell-to-cell faster than other adenoviruses.

The ability of KD1 and KD3 to lyse cells was examined by a trypan blue exclusion cell viability assay which was performed essentially as described by Tollefson et al., *J. Virol.* 70:2296-2306, 1996. In brief, A549 cells were mock-infected or infected with 20 PFU/cell of
10 KD1, KD3, *dI01/07*, *dI327* or *dI309*. At various days p.i., the number of viable cells was determined using a hemocytometer (600 to 1000 cells were counted per time point) and the results are shown in Fig. 4.

Only 25% of the KD1-infected cells and 9% of the KD3-infected cells were alive at 5 days p.i. as compared to 44% of cells infected with *dI01/07*, which has the same E1A
15 mutation as KD1 and KD3. The KD1 and KD3 vectors also lysed cells faster than *dI309*, which has a wild-type E1A region. When infected with *dI327* (ADP⁺, E1A⁺), 94% of the cells were alive after 5 days. When cell lysis was estimated by release of lactate dehydrogenase, KD1 and KD3 once again lysed cells faster than *dI01/07* and *dI309*, and *dI327* caused little cell lysis (data not shown). Thus, ADP is required for efficient cell lysis, and over-expression
20 of ADP increases the rate of cell lysis.

As another means to measure cell lysis and to examine virus replication in cancer cells, separate groups of A549 cells were infected with 20 PFU/cell of KD1, KD3, *dI01/07*, or *dI309* and the amount of intracellular and extracellular virus was determined by plaque assay on A549 cells. At 2 days p.i., the total amount of virus formed in each group was similar, 2-4
25 $\times 10^8$ PFU/ml, indicating that replication of all the viruses is similar. However, when the ratio of extracellular to intracellular virus was calculated, the value for KD1 and KD3 was 2-3 logs higher than for Ad5, *dI309*, or *dI01/07* (data not shown). Thus, virus is released much more rapidly from cells infected with KD1 and KD3, which overexpress ADP, than with viruses expressing wild-type amounts of ADP.

30 The ability of KD1 and KD3 to spread from cell-to-cell was measured in a "cell spreading" assay. In this assay monolayers of A549 cells in a 48 well culture dish were mock-infected or infected with 10^{-3} , 10^{-2} , 10^{-1} , 10^0 , or 10 PFU/cell of *dI327*, *dI309*, Ad5, *dI01/07*, KD1 or KD3. At low PFU/cell, the viruses must go through two or three rounds of replication in order to infect every cell in the monolayer. At 1.0 and 10 PFU/cell, the
35 monolayer should be destroyed by the virus that initially infected the cells. To assess the

amount of spread in the monolayers by 7 days p.i., crystal violet, which stains live cells but not dead cells, was added to the monolayers. The results are shown in Fig. 5.

Remarkably, at 7 days p.i., the monolayer was virtually eliminated by KD1 and KD3 at 10^{-3} PFU/cell, whereas 1.0 PFU/cell was required with *d/01/07*, *d/309* and Ad5. This result attests to the potency of ADP in mediating cell lysis and virus spread in A549 cells. KD1 and KD3 are also more effective than *d/01/07* in killing other types of human cancer cell lines (most purchased from the American Type Culture Collection (ATCC)) as determined in this cell spreading assay. KD1 and/or KD3 killed HeLa (cervical carcinoma), DU145 (prostate), and PC3 (prostate) cells at 10^{-3} PFU/cell, ME-180 (cervix) and Hep3B (liver) at 10^{-1} PFU/cell, and U118 (glioblastoma) and U373 (glioblastoma) at 10 PFU/cell. From 10- to 100-fold more *d/01/07* was required to kill these cells (data not shown). These results indicate that KD1 and KD3 may be effective against many types of cancer.

An important aspect of the finding that ADP overexpressing vectors lyse cells at very low multiplicities of infection is that the multiplicity of infection in human tumors is likely to be low at sites distal to the sight of vector injection or distal to blood vessels that carry the vector to the tumor. Thus, ADP overexpressing vectors have an advantage over vectors that express less ADP or no ADP at all.

Example 3

This example illustrates that KD1 and KD3 replicate poorly in non-growing non-cancerous cells. The replication phenotype of KD1 and KD3 was evaluated using "normal" HEL-299 human fibroblast cells, either growing in 10% serum or rendered quiescent using 0.1% serum. All Ads should replicate well in growing cells, but viruses with the *d/01/07* E1A mutation should do poorly in quiescent cells because E1A is required to drive them out of G_0 . *d/309*, which has wild-type E1A, should replicate well in both growing and growth-arrested cells.

Cells were infected with 100 PFU/cell of KD1, KD3, *d/01/07*, or *d/309*. At different days p.i., virus was extracted and titered. In 10% serum, KD1, KD3, and *d/01/07* replicated well, reaching titers of 10^6 - 10^7 PFU/ml, only slightly less than *d/309* (Fig. 6). However, in quiescent cells, replication of KD1, KD3, and *d/01/07* was 1.5-2 logs lower than in growing cells, ranging from 10^4 to 2×10^5 PFU/ml. The titer of *d/309* reached 10^7 PFU/ml, nearly the level achieved in growing cells. At 10 days p.i., quiescent HEL-299 cell monolayers infected with 100 PFU/cell of KD1, KD3, or *d/01/07* were intact, whereas those infected with *d/309* or *d/327*, which have wild-type E1A, showed strong typical Ad cytopathic effect indicative of cell death (data not shown). Thus, replication of KD1 and KD3 is severely restricted to growing cell lines.

The restriction associated with the *dI01/07* E1A mutation was also tested in primary human cells (purchased from Clonetics) growing as monolayers. Bronchial epithelial cells (Fig. 7) and small airway epithelial cells were not killed by 10 PFU/cell of KD1, KD3, or *dI01/07* at 5 days p.i., whereas they were killed by 10 PFU/cell of *dI309* or *dI327* (data not shown). Lung endothelial cells also were not killed after 10 days by KD1, KD3, or *dI01/07* at 10 PFU/cell, but they were killed by 1 PFU/cell of *dI309*. These monolayers were subconfluent when initially infected, then grew to confluency. The exciting result here is that although these primary cells were growing, they did not support replication in this time frame and were not killed by KD1 or KD3. Thus, it is believed these vectors will be restricted to cancerous cells, and will have little to no effect on cells such as basal cells that are normally dividing in the body. In addition, it is unlikely that KD1 and KD3 will affect dividing leukocytes because such cells are poorly infected by Ad.

In summary, the above experiments demonstrate that KD1 and KD3 lyse cancer cells, spread from cell-to-cell rapidly, and replicate poorly in quiescent and non-cancerous cells. These properties should make them useful in anti-cancer therapy.

Example 4

This example illustrates that KD1 and KD3 inhibit the growth of human tumors in an animal model.

We could not evaluate mouse or rat tumors in normal mice or rats because they are totally non-permissive. Human cancer cell lines growing in nude mice have been used by Onyx Pharmaceuticals (Richmond, CA) to evaluate the efficacy of ONYX-015, an Ad vector lacking expression of the E1B 55 kDa protein (Heise et al., *Nature Med.* 3:639-645, 1997). We have found that A549 cells, which were used in many of our cell culture studies, form excellent rapidly growing solid tumors when injected subcutaneously into nude mice. The average tumor reaches ca. 500 μ l in four weeks, and is encapsulated, vascularized, and attached to the mouse skin (usually) or muscle.

Nude mice were inoculated into each hind flank with 2×10^7 A549 cells. After 1 week tumors had formed, ranging in size from about 20 μ l to 50 μ l. Individual tumors were injected three days later, and at subsequent weeks for 4 weeks (total of 5 injections), with 50 μ l of buffer or 50 μ l of buffer containing 5×10^7 PFU of *dI309*, *dI01/07*, KD1, KD3, or *pm734.1*, with a total virus dose per tumor of 3×10^8 PFU. The mutant *pm734.1* lacks ADP activity due to two nonsense mutations in the gene for ADP, but all other Ad proteins are expected to be synthesized at wild-type levels (Tollefson et al., *J. Virol.* 70:2296-2306, 1996). The efficacy of each virus (or buffer) was tested on six tumors. At weekly intervals, the length (L) and width (W) of tumors were measured using a Mitutoyo digital caliper. Tumor

volumes were calculated by multiplying $L \times W \times W/2$. This value was divided by the tumor volume at the time of the initial virus injection, the fold-increase in tumor growth was calculated, and the average for the six tumors was graphed.

As shown in Fig. 8A, tumors that received buffer continued to grow, increasing about 14-fold by 5 weeks. In contrast, tumors injected with *d/309*, which expresses normal amounts of ADP and lacks the E3 RID and 14.7K and proteins, only grew about 2.5-fold by 5 weeks. With *pm734.1*, which lacks ADP, the tumors grew as well as those that received buffer. Thus, *d/309* markedly decreases the rate of tumor growth, and ADP is required for this decrease. Tumors inoculated with *d/01/07* grew about 8-fold over 5 weeks. Since *d/01/07* is identical to *d/309* except for the E1A mutation, this result indicates that the E1A mutation significantly reduces the ability of Ad to prevent growth of the tumors. This effect is probably due to a reduction in virus replication in the tumors resulting in lower ADP expression, but it could also reflect other properties of E1A in the tumor cells, e.g. the inability of the mutant E1A proteins to induce apoptosis. Most importantly, tumors inoculated with KD1 or KD3 only grew about 2.5-fold. Thus, the overexpression of ADP by KD1 and KD3 allows KD1 and KD3 to reduce tumor growth to a rate markedly slower than *d/01/07* (their parental control virus), and even to a rate similar to that of *d/309*.

The finding that KD1 and KD3 are as effective as wild-type Ad (i.e. *d/309*) in reducing the rate of A549 tumor growth is highly significant in the context of cancer treatment, inasmuch as KD1 and KD3 are restricted to cancer cells whereas wild-type Ad does not have such a restriction.

The tumors in Fig. 8A received five injections of vectors, but only one dose of vector, in this case 5×10^8 of each of KD3 or GZ3, is sufficient to significantly reduce the rate of A549 tumor growth (Fig. 8B).

We have also found that KD1 and KD3 reduce the rate of growth in nude mice of a human liver cancer cell line, Hep3B cells. These cells form rapidly growing tumors that are highly vascularized. Nude mice were inoculated into each hind flank with 1×10^7 of Hep3B cells. After tumors reached about 100 μ l, they were injected twice per week for 3 weeks with 50 μ l of buffer or 5×10^7 PFU of KD1, KD3, or *d/309*. There were typically 8-10 tumors per test virus. The tumor sizes were measured and the fold increase in size at 0 to 3.5 following the initial virus injection was graphed as described above for the A549 tumors. Tumors that received buffer alone grew 9-fold over 3 weeks and were projected to grow about 12-fold over 3.5 weeks (after 3 weeks the mice had to be sacrificed because the tumors were becoming too large) (Fig. 9). Tumors that received KD1 or KD3 grew about 4-fold, establishing that KD1 and KD3 reduce the growth of Hep3B tumors in nude mice. Tumors

that were injected with *d/309* grew 2-fold (Fig. 9). The finding that KD1 and KD3 were somewhat less effective than *d/309* is probably due to the fact that they do not grow as well as *d/309* in Hep3B cells, as indicated by a cell spread assay in culture (data not shown). In any case, the important points are that KD1 and KD3 are effective against the Hep3B tumors, and that they contain the E1A mutation that limits their replication to cancer cells.

These results point to the potency of ADP as an anti-tumor agent when expressed in an Ad vector. It is highly probable that KD1 and KD3 will provide significant clinical benefit when used to infect tumors growing in humans.

Example 5

This example illustrates the use of replication-defective Ad vectors in combination with KD1 or KD3.

It is well established that replication-competent (RC) viruses complement replication-defective (RD) mutants. That is, when the same cell is infected, the competent virus will supply the protein(s) that cannot be made from the mutant genome, and both viruses will grow. To test the ability of KD1 and KD3 to complement RD viruses, two RD vectors expressing β -galactosidase were constructed. The first, named Ad- β -gal, has a cDNA encoding β -gal under the control of the Rous Sarcoma Virus promoter substituted for the deleted E1 region. Ad- β -gal also has the E3 region deleted, including the gene for ADP. The second, named Ad- β -gal/FasL is identical to Ad- β -gal, except that it also expresses murine FasL from the human cytomegalovirus promoter/enhancer. These vectors were constructed by overlap recombination in human 293 cells that constitutively express the Ad E1A and E1B genes and complement replication of the E1-minus vectors.

These RD vectors should infect and express β -gal in A549 cells, but should not replicate because the E1A proteins are lacking. However, the vectors should replicate when cells are co-infected with RC Ads. To prove this, A549 cells were infected with 10 PFU/cell of Ad- β -gal alone, or with 10 PFU/cell of Ad- β -gal plus 10 PFU/cell of KD1, KD3, *d/01/07*, *d/309*, or *d/327*. At 2 days p.i., virus was extracted and Ad- β -gal titers determined by β -gal expression in A549 cells. The yields are shown in Table 2 below.

Table 2

Virus	Yield (blue plaques per ml)
Ad- β -gal	1×10^2
Ad- β -gal + KD1	2×10^5
Ad- β -gal + KD3	3×10^5
Ad- β -gal + <i>dI01/07</i>	4×10^4
Ad- β -gal + <i>dI309</i>	3×10^5
Ad- β -gal + <i>dI327</i>	3.0×10^5

The data in Table 2 indicate that the complementing viruses increased the yield of Ad- β -gal by about 10^3 .

5 A key feature of KD1 and KD3 is that they spread from cell-to-cell faster than other Ads. Accordingly, they should complement the spread of Ad- β -gal. To test this, an infectious center assay was conducted. A549 cells were infected with Ad- β -gal plus KD1, KD3, or *dI01/07*. After 2 h, cells were collected, diluted, and seeded onto monolayers of fresh A549 cells. After 4 days, the cells were stained with X-gal and the results are shown in
10 Fig. 10.

With Ad- β -gal alone, only the originally infected cell (before seeding) should be stained, and the vector should not spread to other cells on the seeded monolayer. This was indeed the case. In monolayers seeded with A549 cells infected with Ad- β -gal alone (dish shown in the top left of Fig. 10A) contained a number of individual blue cells (not visible in the print); examples are shown in the enlarged view Fig. 10B. However, when the
15 monolayers were seeded with A549 cells coinfecting with Ad- β -gal and KD1 or KD3, there were numerous "comets" of blue cells (Fig. 10A). Each comet represents Ad- β -gal which has spread from one initially-infected cell. Most of the cells within a comet were stained with X-gal (Fig. 10C). Comets were also observed with *dI01/07*, but not to the extent of KD1 and
20 KD3 (Fig. 10A). With *dI327* (ADP⁺), there was little spread from the originally infected cell (data not shown). In summary, KD1 and KD3 not only complement the replication of Ad- β -gal, they also enhance its rapid spread.

It is expected that KD1 and KD3 will also complement and enhance the spread of RD vectors expressing anti-cancer therapeutic gene products, and this expectation can be readily

verified using the Ad- β -gal/FasL in replication and infectious center assays as described above.

KD1 and KD3 not only complement the replication of RD vectors in cell culture, they also do so in Hep3B tumors growing in the hind flanks of nude mice. The RD vector used was AdLuc, an Ad that lacks the E1 and E3 regions, and has inserted into the E1 region an expression cassette where the firefly luciferase gene is expressed from the Rous Sarcoma Virus promoter (Harrod et al., *Human Gene Therapy* 9:1885-1898, 1998). The Hep3B tumors were injected with 1×10^7 PFU of AdLuc plus buffer, or 1×10^7 PFU of AdLuc plus 5×10^7 PFU of KD1, KD3, *d*/01/07, or *d*/309. After 2 weeks, mice were sacrificed and tumors excised. Proteins were extracted from the tumors and luciferase activity determined using a luminometer. The luciferase counts per tumor were 6,800 for AdLuc plus buffer, 113,500 for KD1, and 146,900 for KD3 (Fig. 11). Thus, KD3 and KD1 respectively caused a 22-fold and 17-fold increase in luciferase activity. This increase could be due to elevated synthesis of luciferase in cells that were initially coinfecting the AdLuc and KD1 or KD3, and it could also be due to spread of AdLuc from cell to cell in the tumor as mediated by KD1 or KD3.

In summary, infecting a tumor with a replication-competent ADP-overexpressing vector according to the invention together with a RD vector expressing an anti-cancer gene product should greatly increase the amount of anti-cancer protein synthesized in the tumor thereby increasing the ability of the replication-defective vector to promote destruction of the tumor.

Example 6

This example illustrates the construction and characterization of a recombinant Ad vector according to the invention which is replication-restricted to cancerous type II alveolar cells.

As demonstrated above, the *d*/01/07 mutation in KD1 and KD3 limits growth of these vectors to cancer cells. To further restrict their replication phenotype, the E4 promoter in each virus was deleted and replaced by the surfactant protein B (SPB) promoter to produce vectors named KD1-SPB (SEQ ID NO:14), KD3-SPB (SEQ ID NO:15), and *d*/01/07-SPB (SEQ ID NO:16). The SPB promoter is only active in cells containing the TTF1 transcription factor, which has thus far been found primarily in type II alveolar cells of the human lung (Lazzaro et al., *Development* 113:1093-1104, 1991). Thus, KD1-SPB, KD3-SPB, and *d*/01/07-SPB should be severely restricted to cancerous type II alveolar cells of the human lung. Many lung cancers are of this type.

The KD1-SPB and KD3-SPB vectors were prepared as follows. The E4 promoter is located at the right end of the Ad genome (Fig. 1). Using a pCRII-based plasmid (Invitrogen)

containing the Ad5 DNA sequences from the BamHI site (59 map units) to the right hand end of the genome, and using a PCR-based protocol, nearly all the transcription factor binding sites were deleted from the E4 promoter Ad5 base pairs 35,623 to 35,775 and replaced with a 500 base pair fragment containing the SPB promoter (Yan et al., *J. Biol. Chem.* 270:24852-24857, 1995). The final plasmids contain the E4-SPB substitution in the E4 region and the *d/01/07*, KD1, or KD3 versions of the E3 region, respectively, for the viruses *d/01/07*-SPB, KD1-SPB, and KD3-SPB. These plasmids were co-transfected into 293 cells with a fragment containing the left portion of the genome of *d/01/07*, and plaques were allowed to develop. Plaques were screened for the expected features, purified, then expanded into a stock.

- 10 The A549-TTF1 cell line was developed in order to test the prediction that replication of *d/01/07*-SPB, KD1-SPB, and KD3-SPB would be restricted to cancerous cells expressing the TTF1 transcription factor. These cells were co-transfected with two plasmids, one in which TTF1 is expressed from the CMV promoter, and the other coding for resistance to neomycin. Resistant clones were isolated and shown to express TTF1 activity as determined by transient transfection with a plasmid expressing chloramphenicol acetyltransferase from the TTF1-requiring surfactant protein C promoter.

- 20 KD1-SPB and KD1 were subjected to a standard plaque development assay on A549-TTF1 cells and parental A549 cells. The results are shown in Fig. 12. With KD1-SPB on A549 cells, plaques were not visible after 8 days, only about 4% of the final number of plaques were seen after 10 days, and about 50% of final plaques were seen after 12 days. With KD1-SPB on A549-TTF1 cells, plaques were visible after 6 days, and about 60% of plaques were seen after 10 days. Thus, as expected, KD1-SPB grew significantly faster on the cells containing TTF1. KD1 formed plaques more quickly than KD1-SPB on both A549 and A549-TTF1 cells, indicating that the E4 promoter-SPB substitution is not as effective the wild-type E4 promoter in inducing Ad replication. However, this difference between KD1-SPB and KD1 on A549-TTF1 cells is tolerable, with KD1-SPB delayed only about 1 day. Curiously, the final titer obtained for all virus stocks by day 16 was similar, indicating that A549 cells may contain a very small amount of endogenous TTF1 activity. It is predicted that KD3-SPB and *d/01/07*-SPB will behave similarly to KD1-SPB when grown in A549-TTF1 cells and A549 cells.

- 30 The restriction of KD1-SPB to cells containing TTF1 was further examined in a cell spread assay using H441 cells, a TTF1-expressing human pulmonary adenocarcinoma cell line (Yan et al., *supra*), and Hep3B cells, a liver cancer cell line not expected to express TTF1. Culture dish wells containing H441 or Hep3B cells were infected with KD1-SPB or KD1 at multiplicities ranging from 10 to 10⁴ PFU/cell. The H441 and Hep3B cells were

stained with crystal violet at 5 days and 8 days p.i., respectively. KD1-SPB and KD1 grew and spread equally well on H441 cells, causing destruction of the monolayer at 10^{-1} PFU per cell (Fig. 13). (Some of the H441 monolayer has peeled off in the well with KD1-SPB at 10^{-2} PFU per cell, and in the wells with KD1 and KD1-SPB at 10^{-4} PFU per cell; this occasionally occurs in cell spread assays, and it does not reflect virus infection). With Hep3B cells, KD1 grew and spread very much better than KD1-SPB, with 10^{-2} PFU per cell of KD1 causing more destruction of the monolayer as 1.0 PFU per cell of KD1-SPB (Fig. 13).

In summary, this example demonstrates that a replication-competent Ad, which replicates well on cells expressing the appropriate transcription factor, can be constructed with a tissue-specific promoter substituted in place of the E4 promoter. This methodology should be applicable to many other tissue specific and cell type specific promoters. One possibility would be a liver-specific promoter. Another possibility would be to use the E2F promoter, or another promoter with E2F sites, inasmuch as that promoter would be active only in cells such as cancer cells that have free E2F. A third possibility would be to use a regulatable promoter, e.g. the synthetic tetracycline response promoter (Massie et al., *J. Virol.* 72:2289-2296, 1998), where the activity of the promoter is controlled by the level of tetracycline or a tetracyclin analog in the patient.

Example 7

This example illustrates the construction and characterization of vectors which overexpress ADP and are not replication restricted.

As demonstrated above, the *d101/07* E1A mutation in KD1 and KD3 is attenuating, inhibiting growth in non-dividing and even in dividing primary human epithelial and endothelial cells. Ads with this mutation are able to replicate well in dividing cancer cells. However, replication of such E1A mutants is not as efficient as, e.g. *d1309* which has a wild-type E1A gene. For instance, the rate of replication of *d101/07*, as determined by the rate at which plaques develop, is reduced such that *d101/07* plaques appear one day later than those of *d1309* (data not shown). This delay is due in part to a delay in expression of Ad late genes (see Fig. 3). The idea that the *d101/07* mutation retards the rate of replication in A549 cells is further supported by the data in Fig. 8A, where *d101/07* did not prevent tumor growth nearly as well as *d1309*. Despite this negative effect of the *d101/07* E1A mutation, there are theoretical and practical aspects of having this mutation in the KD1 and KD3 vectors, as has been discussed. Nevertheless, one can easily imagine scenarios (e.g. patients with terminal cancer) where the ability of an Ad vector to destroy the tumor supercedes the requirement that the vector be totally restricted to tumor cells. In such cases, it would be advantageous to have vectors similar to KD1 and KD3, but with the wild-type E1A gene. The rates at which such

vectors express their genes, lyse cells, and spread from cell to cell should be higher than those of KD1 and KD3. Such vectors might cause some damage to non-cancerous cells and tissue, but this is also true for other modes of anti-cancer treatment such as surgery, chemotherapy, and radiation therapy.

- 5 In light of these considerations, vectors named GZ1 and GZ3 have been constructed that are identical to KD1 and KD3, respectively, except they have a wild-type E1A region. These vectors were constructed by overlap recombination in A549 cells. The left hand fragment contained the wild-type E1A region of Ad5, and the right end fragment contained the E3 modifications of KD1 or KD3. Plaques were picked, analyzed for the expected
- 10 genotype, plaque-purified, and expanded into CsCl-banded stocks. The titers of these stocks on A549 cells were 2.9×10^{10} PFU/ml for GZ1 and 1.6×10^{11} PFU/ml for GZ3. Thus, these vectors can be grown into high titer stocks comparable to wild-type Ad. The GZ1 and GZ3 plaques are larger and appear much sooner than the plaques for *d1309*. Large rapidly-appearing plaques reflect the ability of Ad to lyse cells and spread from cell-to-cell (Tollefson et al., *J. Virol.* 70:2296-2306, 1996; Tollefson et al., *Virology* 220:152-162, 1996), and this
- 15 property, as discussed, is due to the function of ADP.

- The rate of plaque appearance can be quantitated in a plaque development assay (Tollefson et al., *supra*). Here, a typical plaque assay is performed, and the plaques observed on subsequent days of the assay are calculated as a percentage of the number of plaques
- 20 observed at the end of the plaque assay. As shown in Fig. 14, after 4 days of plaque assay on A549 cells, GZ1 and GZ3 had 48% and 34%, respectively, of the final number of plaques, whereas *d1309* had only 1%. It is very unusual in Ad plaque assays in A549 cells for plaques to appear after only 4 days. These large plaques reflect the overexpression of ADP. These GZ1 and GZ3 plaques appear sooner than those of KD1 and KD3 (data not shown), no doubt
- 25 because GZ1 and GZ3 replicate faster because they have a wild-type E1A region.

- GZ1 and GZ3 lyse cells and spread from cell to cell much more effectively than *d1309*. At 6 days p.i. of A549 cells, approximately as much monolayer destruction was observed with GZ1 and GZ3 at 10^3 PFU per cell as was observed with *d1309* at 10^1 PFU per cell (Fig. 15, top panel). This result further underscores the conclusion that overexpression of
- 30 ADP promotes cell lysis and virus spread.

- In theory, GZ1 and GZ3 should be able to replicate not only in tumor cells but also in normal cells. Although they can replicate in normal cells, it is quite possible that GZ1 and GZ3 may be useful as anti-cancer vectors. First, GZ1 and GZ3 could be injected directly into the tumor. Many tumors are self-contained (encapsulated) except for the blood supply. The
- 35 physical barriers of the tumor could minimize dissemination of the virus to other tissues.

Second, Ads are in general quite benign. Most infections of Ad5 are in infants and result in mild or asymptomatic disease, and are held in check by strong humoral and cellular immunity. Anti-Ad immunity appears to be life-long. GZ1 and GZ3 could be used only in patients who have an intact immune system, and perhaps also with pre-existing anti-Ad immunity. Further, patients could be passively immunized against Ad, using gamma-globulin or even specific purified anti-Ad neutralizing antibodies. Third, considering that Ad5 is a respiratory virus which most efficiently infects lung epithelial cells displaying the specific Ad5 receptor (named CAR) as well as specific integrins (e.g. $\alpha_5\beta_1$), replication-competent vectors derived from Ad5 may not spread efficiently in many non-cancer tissues of the body. In addition, it is believed that versions of GZ1 and GZ3 can be constructed that have the E4 promoter substituted with a tumor-specific, tissue-specific, cell-specific, or synthetic promoter. Such vectors would have the positive features associated with wild-type E1A and ADP, and yet be replication-restricted to tumor tissue and/or to particular cell types.

Example 8

This example illustrates that the combination of KD1, KD3, GZ1, or GZ3 with radiation is more effective in destroying A549 cells, growing in culture or growing as tumors in nude mice, than the vectors alone or radiation alone.

This was shown in a cell spread assay. A549 cells growing in three 48 well culture dishes were mock-infected or infected with different viruses at multiplicities of infection ranging from 10 to 10^4 PFU per cell as indicated in Fig. 15. One dish was not radiated. A second dish received 600 centigrays (cGy) of radiation at 24 h p.i., and a third dish received 2000 cGy of radiation at the same time. All dishes were stained with crystal violet at 6 days p.i. With the cells that were not radiated (top panel in Fig. 15), KD1 and KD3 caused monolayer destruction at lower multiplicities of infection than their parental control, *dl101/07*. This was also true for GZ1 and GZ3 as compared to their parental control *dl309*. (The paucity of cells in the cells infected with GZ1 or GZ3 at 10^4 PFU per cell is an experimental artifact, and is not caused by infection by GZ1 or GZ3). These KD1, KD3, GZ1 and GZ3 results are consistent with earlier results showing that overexpression of ADP leads to increased cell lysis and virus spread.

With the dish that was infected then radiated with 600 cGy there was markedly increased cell killing and virus spread as compared to the non-radiated cells (compare the bottom panel of Fig. 15 with the top panel). For example, with KD1, KD3, GZ1, and GZ3 there was about the same amount of cell destruction in the radiated wells at 10^4 PFU per cell as in the non-radiated wells at 10^3 PFU per cell. Similar results were seen with the dish that

received 2000 cGy of radiation (data not shown), and also with dishes that received 600 or 2000 cGy of radiation 24 h prior to infection (data not shown).

The amount of cell destruction was quantitated by extracting the crystal violet from the cells with 33% acetic acid, then measuring the absorbance at 490 nm (data not shown).

- 5 The absorbance with non-radiated mock-infected cells was set at 100% cell viability. With mock-infected cells that received 600 cGy there was a 15% loss in viability (i.e. 15% less crystal violet was extracted). With KD1 at 10^{-3} PFU per cell, the non-radiated cells were 80% viable whereas the cells receiving 600 cGy of radiation were only about 30% viable. Similar differences in viability between radiated and non-radiated cells were seen with KD3, GZ1, 10 and GZ3. These results argue that the combination of radiation plus vector has a synergistic effect on cell lysis and vector spread, rather than an additive effect. If the effect were only additive, then with the KD1 samples at 10^{-3} PFU per cell, the cell viability should have been 65% (15% reduction in viability due to radiation alone, 20% reduction due to KD1 alone). In fact, the cell viability was 30% rather than 65%.

- 15 As mentioned, approximately as much cell lysis and virus spread were observed with 600 cGy as with 2000 cGy. To determine the optimal dose of radiation to synergize with the vectors, an experiment similar to the one described above was conducted with mock-, d/01/07-, KD1-, KD3-, d/309, GZ1-, or GZ3-infected A549 cells. The 48 well plates received 0, 150, 300, or 600 cGy of radiation at 24 h p.i. Cells were stained with crystal violet. The results with cells receiving 0 versus 600 cGy of radiation were similar to those in Fig. 15. 20 The crystal violet was extracted from the cells infected with 10^{-3} PFU per cell of the difference viruses. The absorbance of crystal violet was determined, and the percent cell viability was graphed, using the absorbance of the non-radiated mock-infected cells as 100% cell viability. As illustrated in Fig. 16, an approximately linear decrease in cell viability in all 25 wells was obtained with increasing radiation dose, although the slope of the line was more negative with KD1, KD3, GZ1, or GZ3 than with mock, d/01/07, or d/309. With KD1, KD3, GZ1, and GZ3, there was much more cell lysis and vector spread with their parental control viruses, and there was synergy between the vectors and radiation. For example, with mock-infected cells, 600 cGy reduced cell viability by about 30% (70% of cells were viable). KD1 30 without radiation reduced cell viability by about 23%. The combination of 600 cGy radiation plus KD1 reduced cell viability to about 85%, more than 53% of which is the sum of radiation alone and KD1 alone. When considering the data in Figs. 15 and 16 together, a dose of about 600 cGy is optimal in this type of cell culture experiment.

- 35 The combination of KD3 or GZ3 with radiation was also examined in the A549 tumor-nude mouse model (see Example 4). A549 cells were injected into the hind flanks of

nude mice, and tumors were allowed to form. When tumors reached approximately 50- μ l, they were injected with buffer or with 5×10^8 PFU of KD3 or GZ3. Eight to ten tumors were injected per test condition. At 1 day p.i., half the mice received 600 cGy of whole body radiation. Tumor size was measured over time, and was plotted as a fold-increase in tumor size versus days p.i. (as described in Example 4). As shown in Fig. 17, the non-radiated buffer-injected tumors grew faster than those injected with KD3 or GZ3. Tumors that received the combination of KD3 and radiation did not grow, and those that received the combination of GZ3 and radiation shrank in size after 14 days. These results indicate that the combination of KD3 plus radiation or GZ3 plus radiation is more effective than either vector alone or radiation alone in reducing the rate of A549 tumor growth in nude mice. It is likely that radiation would increase the effectiveness in treating tumors of KD1 and GZ1, or indeed any other replication-competent or replication-defective Ad vector.

The mechanism by which radiation causes the ADP overexpressing vectors to lyse cells and spread from cell-to-cell more effectively is not understood. Radiation is expected to induce cellular DNA repair mechanisms, and that may allow for more efficient synthesis of Ad DNA. Radiation may enhance the function of ADP. ADP probably functions by interacting with one or more cellular proteins, and radiation may affect this protein(s) such that ADP functions more efficiently.

It is believed that KD1, KD3, GZ1, or GZ3, or any other replication-competent Ad vector, when used in combination with radiation, will be more effective than vector alone or radiation alone in providing clinical benefit to patients with cancer. The vectors should allow more tumor destruction with a given amount of radiation. Stated another way, radiation should cause more tumor destruction with a given amount of vector. These vectors should also allow the radiation oncologist to use less radiation to achieve the same amount of tumor destruction. Less radiation would reduce the side effects of the radiation.

It is also believed that a cocktail of vectors when used in combination with radiation will be more effective than the cocktail alone or radiation alone. The cocktail could consist of ADP producing vectors plus one or more replication defective vectors expressing an anticancer therapeutic protein (see Example 5).

Example 9

This example illustrates a structure-function analysis of adenovirus death protein.

ADP is an 11.6 kDa N-linked O-linked integral membrane glycoprotein that localizes to the inner nuclear membrane (NM) (Scaria et al., Virology 191:743-753). As illustrated in Fig. 18, the Ad2-encoded ADP (SEQ ID NO:6) consists of 101 amino acids; aa 1-40 (SEQ ID NO:17) are luminal, aa 41-59 (SEQ ID NO:18) constitute the transmembrane signal-anchor

(SA) domain, aa 63-70 (SEQ ID NO:19) constitute a basic proline (BP) domain within the nucleoplasmic (NP) domain, which constitutes aa 61-101 (SEQ ID NO:20). To determine which domains in ADP are required to promote cell death, a number of deletion mutants of *rec700* were prepared which lacked various portions of the ADP gene and examined for the ability of ADP to localize to the NM and promote death. The *rec700* virus is an Ad5-Ad-Ad5 recombinant, which has been described elsewhere (Wold et al., *Virology* 148:168-180, 1986).

The structure of ADP in *rec700* and in each deletion mutant is schematically illustrated in Fig. 18. The ADP gene in each deletion mutant has been sequenced using PCR methods to insure that the mutations are correct. The structure and activity of ADP in the deletion mutants was tested by infecting A549 cells followed by immunoblot analysis of the ADP mutant proteins as well as the ability to lyse cells. All deletion mutants expressed a stable ADP protein except *pm734.1* ($\Delta 1-48$, i.e. aa 1-48 are deleted). The *pm734.7* (N₁₄) ADP, which has Asn₁₄ mutated to Ser, is O-glycosylated but not N-glycosylated because Asn₁₄ is the only N-glycosylation site (data not shown). The *dl735* ($\Delta 4-11$) ADP is N-glycosylated but not O-glycosylated because the sites for O-glycosylation are deleted (data not shown). The *pm734.4* (M56) ADP, which has Met₅₆ in the SA domain mutated to Ser, contains exclusively N-linked high-mannose oligosaccharides (data not shown); this occurs because the Met₅₆ mutation precludes exit of ADP from the endoplasmic reticulum (ER). The *dl738* ADP, which lacks aa 46-60 in the signal-anchor domain, forms insoluble aggregates in the cytoplasm; therefore, aa 41-59 do in fact include the signal-anchor domain. The *pm734* ($\Delta 1-40$) ADP, which initiates at Met₄₁ at the N-terminus of the SA domain, comigrated with the lower group of bands generated by proteolytic processing (data not shown). This indicates that the proteolytic cleavage sites occur near Met₄₁. Consistent with this, the proteolytic products were not seen with *dl737* ($\Delta 29-45$) (data not shown). Also, the size of the products decreased in all mutants with deletions within aa 41-101 (*dl715.1*, *dl715*, *dl714*, *dl716*) (data not shown).

The ability of these mutants to promote cell death was monitored by trypan blue exclusion, plaque development, and lactate dehydrogenase release assays (Tollefson et al., *J. Virol.* 70:2296-2306, 1996). The trypan blue results in Fig. 15A indicate that the death-promoting function of ADP was abolished by deletion of aa 1-40 (*pm734*), aa 11-26 (*dl736.1*), aa 18-22 (*dl735.1*), or aa 4-11 (*dl735*). Mutation of the N-glycosylation site at Asn₁₄ (*pm734.7*) reduced the death-promoting activity to about 50% of *rec700* (WT). *dl737* ($\Delta 29-45$) was efficient as *rec700* in promoting cell death; this indicates that the proteolytic processing products must not be required to promote cell death because they are not formed with *dl737*. The SA domain is essential for death because *dl738* ($\Delta 46-60$) and *pm734.4*

(M56) were completely defective (Fig. 19). *dl715.1* was nearly completely defective, indicating that the BP domain is extremely important. Surprisingly, aa 71-94 (*dl714*), 76-89 (*dl715*), and 79-101 (*dl716*) could be deleted without affecting the death-promoting activity of ADP (Fig. 19). On the other hand, deletion of aa 81-88 (*dl717*) nearly completely
 5 abolished the activity of ADP (Fig. 19); this is probably the result of aberrant sorting of ADP (see below). Similar results were obtained when the ability of these ADP mutants to promote cell death was examined with standard plaque development, LDH-release and MTT assays.

The effects of these mutations on the intracellular localization of ADP are extremely interesting. When examined by immunofluorescence (IF) at 33 h p.i. (data not shown), ADP
 10 from *rec700* (WT) localized crisply to the NM; localization to the Golgi was also apparent. With *dl714* ($\Delta 71-94$) and *dl715* ($\Delta 76-89$), ADP localized to all membranes, i.e. the ER, Golgi, plasma membrane, and NM. This was even more apparent at 45 h p.i. (data not shown). Thus, aa 71-94 appear to include a signal that directs ADP specifically to the NM. ADP is very likely sorted from the *trans*-Golgi network (TGN) to the NM, so this putative signal in
 15 ADP probably functions in this sorting pathway. ADP from *dl717* ($\Delta 81-88$) is intriguing: it localized to the NM and Golgi, but in many cells "dots" and circular structures were observed. Again, this was more apparent at 45 h p.i. when these structures were the prominent feature. *dl717*-infected cells have not begun to die at 45 h p.i., so these structures are not cellular remnants. The intriguing possibility is that these structures are membrane vesicles that have
 20 pinched off from the TGN but are defective in targeting to and/or fusing with the NM.

With *dl738* ($\Delta 46-60$ in the SA domain), ADP aggregated in the cytoplasm. This again indicates that aa 46-60 include the SA sequence. With *pm734.4* (M56), ADP localized primarily to the NM. As discussed above, the *pm734.4* ADP has exclusively high-mannose N-linked oligosaccharides, indicating that it never leaves the ER. Perhaps the putative NM-
 25 localization signal in the C-terminal region of the *pm734.4* ADP targets ADP to the NM by lateral diffusion from the ER (which is continuous with the outer and inner NM).

With *dl737* ($\Delta 29-45$), ADP localized to the NM. ADP from *pm734* ($\Delta 1-40$), *pm734.7* (N14) (N-linked glycosylation cannot occur), and *dl735* ($\Delta 4-11$; the O-glycosylation sites are deleted) localized much more prominently to the Golgi than the NM. ADP from *dl735.1*
 30 ($\Delta 18-22$) and *dl736.1* ($\Delta 11-26$) also localized much more strongly to the Golgi than the NM. Thus, residues 1-26 and/or glycosylation appear to be required for efficient transport of ADP from the Golgi/TGN to the NM.

In summary, aa 41-59 include the SA domain, Met₅₆ in the SA domain is required for exit from the ER, aa 1-26 are required for efficient exit from the Golgi, and aa 76-94 are
 35 required to target ADP specifically to the NM. With respect to promoting cell death, the

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essential regions are aa 1-26, the SA domain (ADP does not enter membranes), Met₅₆ in the SA domain, and the BP domain (aa 63-70). It is not clear whether the defective death-promoting phenotype of *pm734* (Δ 1-40), *dl735* (Δ 4-11), *dl735.1* (Δ 18-22), *dl736.1* (Δ 11-26), and *pm734.7* (N14) is due to lack of sequences (or oligosaccharides) that promote death or to much slower exit of ADP from the Golgi to the NM. *dl714* (Δ 71-94) and *dl715* (Δ 76-89) express a wild-type phenotype for promoting death even though they are defective in localizing specifically to the NM; this is probably because sufficient ADP still enters the NM to promote death. Even though the deletion in *dl717* (Δ 81-88) lies within the deletions in *dl715* (Δ 76-89) and *dl714* (Δ 71-94), the *dl717* ADP is only about 15% as efficient as *rec700* (WT), *dl715* and *dl714* in promoting death. This may be because the *dl717* ADP tends to remain in vesicles rather than localizing to the NM. Altogether, these data indicate that ADP must localize to the NM in order to promote cell death.

Example 10

This example further characterizes the tissue specific Ad vectors described in Example 6. As discussed therein, the Ad E4 promoter is deleted and replaced with the promoter for surfactant protein B (SPB) in these vectors (Figure 24).

Materials and Methods

Cells, vectors and methods described in Example 6 were also used in this Example. In addition to the human cancer cell lines A549 (human lung carcinoma), Hep 3B (human hepatocellular carcinoma), and H441 (papillary lung adenocarcinoma) used in Example 6, HEK 293 cells (obtained from Microbix (Toronto, ON)) and VK10-9 cells were used. VK10-9 cells are 293 cells that in addition to E1 contain and express E4 and pIX. These cells will be referred to as 293-E4 cells.

Experiments employing phase contrast microscopy of Hep 3B and H441 cells were performed as follows. Monolayers of Hep 3B or H441 cells were grown in 60 mm dishes with 5 ml of DMEM (10% FBS), and were mock-infected or infected with KD1 or KD1-SPB at a multiplicity of infection of 10 plaque forming units (PFU) per cell. Phase contrast photographs of monolayers were taken at 4 and 7 days postinfection (p.i.).

Experiments employing western blots of H441 or Hep 3B cells were performed as follows. H441 or Hep 3B cells (in 60 mm dishes) were infected with 10 PFU/cell of KD1 or KD1-SPB. At 24 h p.i., the cells were washed three times with PBS and harvested by scraping. The cells were lysed by RIPA buffer. The protein concentration was measured by the BIO-RAD DC Protein Assay Kit (BIO-RAD Laboratories, Hercules, CA) and 10 μ g of each sample were electrophoresed on 15% sodium dodecylsulfate polyacrylamide gels (SDS-PAGE). The gels were electroblotted onto PVDF membranes (Immobilon, Millipore,

Bedford, MA). The membranes were blocked in TBST (50 mM Tris-Cl, pH 7.6, 150 mM NaCl, 0.2% Tween 20) containing 10% dry milk (Carnation) overnight at 4°C. After blocking, the membranes were incubated with a rabbit polyclonal antiserum against E4ORF3 (gift of Gary Ketner) or ADP (Tollefson et al., *J. Virol.* 66:3633-3642, 1992), or with M73, a
 5 monoclonal antibody against E1A (Harlow et al., *J. Virol.* 55:533-546, 1985). The secondary antibodies were goat anti-rabbit IgG-HRP or goat anti-mouse IgG-HRP. The blots were developed using the ECL protocol (Amersham Pharmacia, Arlington Heights, IL).

Experiments employing a lactate dehydrogenase release assay for cell lysis (Tollefson et al., *J. Virol.* 70:2296-2306) were performed as follows. H441 cells (7.7×10^5 cells per 35 mm dish) and Hep 3B cells (9.0×10^5 cells per 35 mm dish) were infected at 20 PFU/cell in one ml serum-free DMEM. After an adsorption period of 1 h, 3 ml of DMEM (10% FBS) were added (final FBS concentration of 7.5%). Cells were incubated at 37°C with 6% CO₂. At daily intervals, supernatants were collected, microfuged to remove floating cells, and cell-free supernatants were frozen at -70°C until assayed. Total lysis samples were prepared by
 10 addition of 10X lysis buffer included in the Cyto Tox 96 kit (Promega, Madison, WI). After all samples were collected, 20 µl samples were assayed in triplicate using the LDH assay kit Cyto Tox 96 and read on an EL340 Microplate reader (BioTec™ Instruments, Inc.) at 490 nm.

Experiments employing immunofluorescence evaluation of H441 and Hep 3B cells were performed as follows. H441 and Hep 3B cells were plated on Corning #1 coverslips in 35 mm dishes. H441 (1.5×10^6 cells/35 mm dish) and Hep 3B (9.0×10^5 cells/35 mm dish) were infected with 20 PFU/cell of the indicated viruses in 1 ml serum-free DMEM. After 1 h, 1 ml of DMEM/20% FBS was added (final concentration of 10% FBS). At the indicated times (48 h or 6 d p.i.), cells were fixed for 10 min in 3.7% paraformaldehyde in PBS, then
 25 permeabilized for 6 min in methanol (-20°C) and rehydrated in PBS. Coverslips were stained with rabbit anti-peptide antiserum against the Ad E2A-coded DNA binding protein (DBP) (1:400 dilution; gift of Maurice Green) and mouse monoclonal antibody against fiber (1:400 dilution; gift of Jeff Engler) or were stained with rabbit antiserum to E4ORF3 (1:250 dilution; gift of Gary Ketner). Secondary antibodies (Cappel/ICN) were used at 1:50 dilution. All
 30 antibodies were diluted in PBS containing 1% BSA and 0.1% sodium azide. Photographs were taken on a Nikon epifluorescence microscope using a 100X Planapo lens and Tmax 400 film (Kodak). The film was developed in Diafine developer.

Analysis of viral DNA replication by Southern hybridization was performed as follows. H441 and Hep 3B cells were grown in 60 mm dishes in DMEM supplemented with
 35 10% FBS. Cells were infected at 70% confluence with 10 PFU/cell of KD1 or KD1-SPB.

Dishes were incubated in humidified 5% CO₂ atmosphere at 37°C. Total genomic DNAs were isolated at 5, 24, 48, 72, and 96 h p.i. Equal amounts of total genomic DNAs were digested with HindIII and resolved on a 1% agarose gel prior to transfer onto membranes. A random primer ³²P-labeled pBHG10 plasmid probe (Bett et al., *Proc. Natl. Acad. Sci. USA* 91:8802-8806, 1994) was used for hybridization, and the blots were autoradiographed. DNA fragments were quantitated on a Molecular Dynamics PhosphorImager.

Virus yields were determined as follows. Hep 3B cells or H441 cells grown as monolayers in 35 mm dishes were infected with 10 PFU/cell of KD1 or KD1-SPB. At days 0 to 4 (for H441) or days 0 to 9 (for Hep 3B) p.i., cells and culture medium were frozen at -70°C. Samples were frozen and thawed three times to release the virus from the cells, and total virus yields were determined by plaque assay on A549 monolayers.

The effect of KD1-SPB and KD1 on H441 and Hep 3B tumors was examined in a nude mouse model (Doronin et al., *J. Virol.* 74:6147-6155, 2000). Tumor cells (10⁷ cells in 200 µl of DMEM, 50% Matrigel [Becton Dickinson Labware, Bedford, MA] for H441 cells, or 10⁷ cells in 200 µl of DMEM plus 10% Matrigel for Hep 3B cells) were injected into flanks of 5-6 weeks old athymic nude mice and allowed to grow for three weeks to about 100 µl (H441) or 150 µl (Hep 3B) volumes. Pre-established tumors (n = 10) were injected with 50 µl of DMEM or 5 x 10⁷ PFU of indicated viruses in DMEM. Injections of the viruses were repeated twice weekly for 3 weeks to the total dose of 3.0 x 10⁸ PFU per tumor. Tumor size measurements were taken twice per week for H441 cells, or weekly for Hep 3B cells using a Sylvac digital caliper. Tumor volumes were calculated in according to the formula: length x width² / 2. Data are represented as means of increase in tumor size relative to the tumor size at the initial injection.

Results

The properties of KD1-SPB in various cell types were compared to those of its "parent", KD1. Figure 25 shows the plaque development properties of these vectors on 293-E4, 293, and A549 cells. The data are plotted as the number of plaques seen on any day of the plaque assay as a percentage of the number of plaques seen at the end of the assay (i.e. when new plaques cease to appear) (Tollefson et al., *J. Virol.* 70:2296-2306, 1966). This assay is an indicator of the size of the plaques. KD1 formed plaques equally well on 293-E4 and 293 cells (Figure 25A). With KD1-SPB, plaques were observed about 3-4 days sooner on 293-E4 compared to 293 cells (Fig. 2A). On A549 cells, KD1 formed plaques 4-6 days sooner than KD1-SPB (Figure 25B).

The properties of KD1-SPB versus KD1 were characterized in detail in H441 cells, a human papillary lung adenocarcinoma cell line known to express the TTF1 transcription

factor and in which the SPB promoter is active (Yan et al., *J. Biol. Chem.* 270:24852-24857, 1995). Hep 3B cells, a human hepatocellular carcinoma in which the SPB promoter should not be active, were used as a negative control. H441 and Hep 3B monolayers were infected with 10 PFU/cell of KD1 or KD1-SPB and photographed at 4 and 7 days p.i. Mock-infected

5 Hep 3B cells formed a relatively homogeneous monolayer, but H441 cells tended to form structures that resemble syncytia (Figure 26A, B). As expected, KD1 produced cytopathic effect (CPE) on both cell lines at 4 and 7 days p.i. (Figure 26A, B). Also as expected, KD1-SPB caused CPE on H441 cells but not on Hep 3B cells. Since CPE in Ad-infected cells is usually an indicator of virus growth, these results suggest that KD1-SPB grows in H441 but

10 not in Hep 3B cells.

To examine viral DNA replication, H441 and Hep 3B cells were infected with 10 PFU/cell of KD1 or KD1-SPB, then the accumulation of viral DNA was determined by DNA blot. With H441 cells, KD1 and KD1-SPB DNAs were readily detected at similar levels at 48-96 h p.i. (Figure 27A). With Hep 3B cells, KD1 DNA levels were similar to those in

15 H441 cells, but KD1-SPB DNA was barely detectable. This was confirmed by Phosphorimager analysis of the DNA bands (Figure 27B).

Growth of KD1-SPB and KD1 in H441 and Hep 3B cells was determined by a single step growth assay. Cells were infected with 10 PFU/cell of vector, then total vector yield was determined by plaque assay. Total yield of both vectors was similar in H441 cells, reaching a

20 plateau after 2 days (Fig. 28A). KD1 yield plateaued in Hep 3B cells after 2-4 days p.i. (Figure 28B). However, KD1-SPB levels were about 5 logs lower in Hep 3B cells after 2-4 days, and even by 9 days they had not achieved the levels of KD1. We conclude that KD1-SPB grows with significant specificity on H441 versus Hep 3B cells. Further, KD1-SPB grows as well as KD1 on H441 cells, indicating that the E4 promoter deletion by itself does

25 not significantly compromise the vector, and that the E4 promoter can be replaced by a tissue-specific promoter in a replication-competent vector.

To obtain further details on the replication of KD1-SPB vs KD1 in H441 and Hep 3B cells, the expression of representative Ad proteins by KD1-SPB and KD1 was examined. H441 or Hep 3B cells were mock-infected or infected with 10 PFU/ml of KD1 or KD1-SPB,

30 then at 24 h p.i. the proteins were extracted and the E1A, E4ORF3, and ADP proteins were examined by immunoblot. E4ORF3 is one of the six proteins coded by the E4 transcription unit (Leppard, *J. Gen. Virol.* 78:2131-2138, 1997). As anticipated, KD1-SPB expressed E4ORF3 well in H441 cells, but only at trace levels in Hep 3B cells (Figure 29). KD1-SPB expressed the E1A proteins in Hep 3B cells. Synthesis of E1A proteins by KD1-SPB in Hep

35 3B cells is expected because E1A expression does not require E4 proteins; it also indicates

that the block to infection with KD1-SPB is downstream of E1A. KD1 expressed E1A in both cell lines, but the amount was less than obtained with KD1-SPB in Hep 3B cells (Figure 29). The increased E1A levels seen with KD1-SPB may reflect its poor ability to enter the late phase of infection (see Discussion). KD1-SPB expressed ADP as well as KD1 in H441

5 cells, but it did not make detectable ADP in Hep 3B cells. ADP is primarily a late protein, so this result is consistent with the relative lack of E4 protein expression, DNA replication, and growth of KD1-SPB in Hep 3B cells.

To gain insights into replication events that occur in individual cells, expression of E4ORF3, the E2A-DBP, and the fiber late protein was examined by immunofluorescence.

10 H441 or Hep 3B cells were infected with 20 PFU/cell. At 48 h or 6 days p.i., cells were fixed and immunostained. E4ORF3 was detected in the nuclei of H441 cells at 48 h p.i. with KD1, KD1-SPB, or dl309 (Figure 30A). (dl309 is an Ad5 mutant that has wild-type E1A, expresses Ad5 levels of ADP, and lacks the E3-RID and E3-14.7K genes). E4ORF3 could not be detected in the vast majority of Hep 3B cells infected with KD1-SPB (Figure 30A), even at 6

15 days p.i. (Figure 30B). Thus, KD1-SPB expresses E4ORF3 well in H441 but not in Hep 3B cells.

Figure 31A shows double label immunofluorescence of DBP and fiber in the same Hep 3B cells at 48 h p.i. with KD1 or KD1-SPB. With KD1, there was a strong speckled staining pattern in the nucleus that is typical for DBP at 48 h p.i. (Figure 31A, top left panel).

20 There was strong staining of fiber throughout these same cells (Figure 31A, top right panel). Staining of the cytoplasm and nucleus is expected because fiber is synthesized in the cytoplasm and then transported to the nucleus where virions assemble. With KD1-SPB at 48 h p.i., about 25% of the cells showed the speckled staining for DBP, and only one cell (7% of total) with the advanced speckled pattern was also stained for fiber (Figure 31A, bottom two

25 panels). Even at 6 days p.i., only about 30% of cells showed staining for DBP, and about 20% for fiber (Figure 31B). Thus, markedly fewer Hep 3B cells infected with KD1-SPB expressed DBP and especially fiber as compared to KD1. These results indicate that KD1-SPB replicates as well as KD1 in H441 cells, no doubt because the SPB promoter is active in H441 cells (Yan et al., *J. Biol. Chem.* 270:24852-24857, 1995). KD1-SPB barely replicates

30 in Hep 3B cells, presumably because the SPB promoter is minimally active in these cells.

At the culmination of replication, Ad-infected cells are lysed and the virus spreads to other cells; this process is mediated in large part by ADP (Tollefson et al., *Virology* 220:152-162, 1996; Tollefson et al., *J. Virol.* 70:2296-2306, 1996). To examine vector-induced cell

35 lysis, H441 and Hep 3B cells were mock-infected or infected with 20 PFU/cell of KD1, KD1-SPB, or dl309, and cell lysis was determined by release of lactate dehydrogenase (Tollefson et

al., *J. Virol.* 70:2296-2306, 1996). All vectors lysed H441 cells beginning at 2-3 days p.i. (Figure 32A). KD1 and dl309 also lysed Hep 3B cells in the same time period; however, KD1-SPB caused only minimal cell lysis (Figure 9B). Thus, these data, along with the cell spread data in Example 6 and Figure 13, demonstrate that KD1-SPB lyses cells and spreads efficiently from cell-to-cell in H441 but not Hep 3B cells.

An experiment was conducted to determine whether KD1-SPB or KD1 would suppress H441 tumors in nude mice. H441 cells were injected into each hind flank. When tumors had grown to about 100 μ l (H441) or 150 μ l (Hep 3B), they were injected twice weekly for 3 weeks with DMEM (mock) or 5×10^7 PFU of test virus in 50 μ l of DMEM (3.0×10^8 total PFU). Ten tumors (5 mice) were used for each virus. Growth of H441 tumors was suppressed similarly by KD1-SPB and KD1 (Figure 33A). KD1 suppressed growth of Hep 3B tumors, whereas KD1-SPB caused only minimal suppression (Figure 33B). These results show that KD1-SPB is as effective as KD1 in suppressing tumors when the SPB promoter is active. Further, the cell type specificity observed with KD1-SPB in vitro is maintained in vivo.

Discussion

Tumor specificity is one of the biggest challenges facing cancer gene therapy, i.e. having the therapeutic gene be expressed specifically in cancer cells. Specificity is very important for RC viruses. Two main strategies have been described that in theory confer specificity: transductional targeting and transcriptional targeting. Directing specificity of vectors toward specific cell surface receptors on the target cells has been attempted through various methods. Although this approach is theoretically attractive it might encounter multiple obstacles such as the lack of incorporation of the engineered protein into the virion (Scaria et al., *Virology* 191:743-753, 1992) or lack of infectivity through the targeted receptor (Cosset et al., *J. Virol.* 69:6314-6322, 1995). Transcriptional targeting utilizes tumor and tissue specific promoters. In replication-defective vectors these regulatory sequences confine the expression of cytotoxic genes to specific tissues. In replication-competent vectors, as an added layer of regulation, vector replication per se can be placed under the control of tumor or tissue specific promoter/enhancer sequences. In replication-competent Ad, insertion of the tissue or tumor specific promoter/enhancer into the E1A promoter/enhancer region has been used exclusively (Hallenbeck et al., *Hum. Gene Ther.* 10:1721-1733, 1999; Rodriguez et al., *Cancer Res.* 57:2559-2563, 1997; Yu et al., *Cancer Res.* 59, 4200-4203, 1999; Yu et al., *Cancer Res.* 59:1498-1504, 1999). The rationale behind these vectors is that expression of E1A and therefore the whole Ad transcription program will depend on these tissue or tumor specific promoters. However, as a generic approach, there may be difficulties. The E1A

enhancer/promoter is very complex. The enhancer controls not only the E1A promoter but also distant promoters such as the E4 promoter (Shenk, T. pp. 2111-2148 *In* B.N. Fields, D.M. Knipe, and P.M. Howley (eds.), *Fields Virology*, Lippincott-Raven, Philadelphia, 1996). In addition, it has been shown that the E1A enhancer in the inverted terminal repeat
5 region changes tissue specificity of cellular promoters (Shi et al., *Hum. Gene Ther.* 8:403-410, 1997). Also, the E1A enhancer/promoter is partially embedded within the signals required to package the Ad genome into virions, and it may be problematic to remove all the E1A enhancer elements without impairing virus production. Accordingly, we chose to replace the E4 promoter with a tissue specific promoter. E4 genes are essential for Ad
10 replication, and therefore we expected that the replication of the recombinant virus would be dependent on the tissue specific regulatory elements.

To construct KD1-SPB, the ca. 300 bp of the E4 promoter was deleted and the B-500 version (ca. 500 bp) of SPB promoter was inserted (Yan et al., *supra*) (Figure 24 C, D). We selected the SPB promoter because of its strict tissue specificity: it is exclusively active in
15 type II alveolar cells and bronchial epithelial cells of the lung (Bohinski et al., 1994, *Mol. Cell. Biol.* 14:5671-5681, 1994). Since the parental virus KD1 contains and expresses two E1A mutations that restrict virus replication to tumor cells (Doronin et al., *supra*), we anticipated that the virus would selectively replicate in cells derived from lung tumors. Thus, H441 cells, a papillary lung carcinoma cell line, were used to characterize the replication,
20 gene expression, and functional profile of KD1-SPB.

KD1-SPB formed plaques 3-4 days sooner on 293-E4 cells that express E4 proteins than on 293 cells, whereas KD1 formed plaques with the same kinetics on both cell lines. These data show that the E4 promoter is active in 293 cells, and that the SPB promoter displays very low activity in 293 cells. It is not clear why KD1-SPB forms plaques on 293
25 cells; these cells are derived from human embryonic kidney and at least one of the transcription factors regulating the SPB promoter (Bohinski et al., *supra*), hepatocyte nuclear factor 3, is expressed in embryonic kidney. It is also possible that TTF1, the master regulatory factor of SPB expression, is minimally active in 293 cells.

KD1 grew to equally high titers in H441 and Hep 3B cells (Figure 28A, B). In
30 contrast, KD1-SPB replicated as efficiently as KD1 in H441 cells, in which the SPB promoter is active (Yan et al., *supra*) (Figure 28A), but replicated poorly in Hep 3B cells, most likely because the SPB promoter is inactive (Figure 28B). This selectivity has been confirmed by measuring viral DNA production in the two cell lines. KD1-SPB DNA replication was similar both kinetically and quantitatively to KD1 DNA replication in H441, however in Hep

3B cells, KD1-SPB DNA was almost undetectable (Figure 27A, B). The cytopathic effect, a surrogate marker of Ad replication, showed a similar specificity (Figure 26).

To further confirm our predictions on the molecular basis of the observed tissue specificity we monitored viral protein expression. When cells were infected with KD1-SPB all the viral proteins early or late, except for E1A, were expressed in a tissue-specific fashion (high expression in H441, low to undetectable expression in Hep 3B) (Figures 29-31). We found a good correlation between the levels of E4 promoter activity (E4ORF3 expression) and the expression of E2A-DBP, ADP, and fiber proteins. Thus, the SPB promoter retains its tissue specificity in the Ad genome and it seems to be the limiting factor of Ad gene expression in the cell lines tested. As expected, expression of E1A is not tissue-specific. Thus, the regulatory step of tissue-specific Ad DNA replication is downstream of E1A. In Hep 3B cells, KD1-SPB expressed E1A at a higher level than did KD1 (Figure 29), strongly suggesting that KD1-SPB replication in most of the Hep3B cells remains at the early stage.

The cytolytic effect of KD1-SPB also showed a tissue-specific profile (Figure 32; Figure 13 of Example 6), i.e., preferential lysis of H441 cells over Hep 3B cells, a pattern similar to the specificity observed at the level of DNA replication (Figure 27) and viral protein synthesis (Figures 29-31). This cell type specificity was also observed when these cells were growing as tumors in nude mice. Growth of H441 tumors was suppressed by KD1-SPB and KD1 at similar efficacy (Figure 33A). In contrast, KD1-SPB unlike KD1 had only minimal effect on the growth of Hep 3B tumors (Figure 33B).

In summary, substitution of the E4 promoter with a tissue specific promoter allows highly tissue specific replication of Ad vectors and in the target tissue it is as efficient as the replication of the parental virus. KD1-SPB lacks all E3 genes except ADP. E3 gp19K, RID and 14.7K have been shown to protect Ad-infected cells from attack by cytotoxic lymphocytes and apoptosis-inducing cytokines such as tumor necrosis factor and Fas ligand (Wold et al., pp. 200-232 *In A.J. Cann (ed.), DNA Virus Replication: Frontiers in Molecular Biology*, Oxford University Press, Oxford, 2000; Wold et al., *Curr. Opin. Immunol.* 11:380-386, 1999).

The therapeutic index (virus produced in H441 cells compared to Hep 3B cells) of KD1-SPB is 10^4 - 10^5 for the first 4-5 days (Figure 28). These data compare to data reported by Calydon (10^4 - 10^5) for their prostate specific viruses (Rodriguez et al., *supra*; Yu et al., *Cancer Res.* 59, 4200-4203, 1999; Yu et al., *Cancer Res.* 59:1498-1504, 1999). We suggest that KD1-SPB has some added advantage over vectors reported by other laboratories because it encodes a mutant form of E1A that restricts replication to cancer cells (Doronin et al., *supra*).

Although the lung ranks as the second highest cancer site for both men and women in the U.S. Reis et al., *Cancer Res.* 88:2398-2424, 2000), lung cancer has not been a major target for cancer vector gene therapy since intratumoral injection of virus is generally not feasible in the lungs. However, there has been a recent report of intratumor injection of a replication-defective Ad vector into a lung tumor, and such an approach could be attempted with KD1-SPB. It may also be feasible to administer KD1-SPB systemically in the lung.

In view of the above, it will be seen that the several advantages of the invention are achieved and other advantageous results attained.

As various changes could be made in the above methods and compositions without departing from the scope of the invention, it is intended that all matter contained in the above description and shown in the accompanying drawings shall be interpreted as illustrative and not in a limiting sense.

All references cited in this specification, including patents and patent applications, are hereby incorporated by reference. The discussion of references herein is intended merely to summarize the assertions made by their authors and no admission is made that any reference constitutes prior art. Applicants reserve the right to challenge the accuracy and pertinence of the cited references.

EDITORIAL NOTE

APPLICATION NUMBER - 13244/01

The following Sequence Listing pages 1 to 75 are part of the description. The claims pages follow on pages "51" to "52".

SEQUENCE LISTING

<110> Wold, William S.M.
Toth, Karoly
Doronin, Konstantin
Tollefson, Ann E.

<120> Replication-Competent Anti-Cancer Vectors

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<151> 1999-07-12

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Leu Gly Met Trp Trp Phe Ser Ile Ala Leu Met Phe Val Cys Leu Ile
35         40         45

Ile Met Trp Leu Ser Cys Cys Leu Lys Arg Lys Arg Ala Arg Pro Pro
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Met Phe Val Cys Leu Ile Ile Met Trp Leu Ile Cys Cys Leu Lys Arg
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Arg Arg Ala Arg Pro Pro Ile Tyr Arg Pro Ile Ile Val Leu Asn Pro
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 20 25 30
 Val Asn Asp Trp Ala Ser Leu Asp
 35 40

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 Trp Leu Ile

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<400> 19
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 1 5

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 Ile Val Leu Asn Pro His Asn Glu Lys Ile His Arg Leu Asp Gly Leu
 20 25 30
 Lys Pro Cys Ser Leu Leu Leu Gln Tyr Asp
 35 40

THE CLAIMS DEFINING THE INVENTION ARE AS FOLLOWS:

1. A recombinant adenovirus which overexpresses an adenovirus death protein and which is replication-competent in neoplastic cells, wherein said adenovirus death protein
5 comprises a sequence selected from a sequence as set forth in SEQ ID NO:5, SEQ ID NO:7, SEQ ID NO:8 SEQ ID NO:10, SEQ ID NO:11 or SEQ ID NO:12, or a conservatively substituted variant thereof.

2. A recombinant adenovirus according to claim 1 which is further
10 characterized by lacking expression of at least one E3 protein selected from the group consisting of: gp19K; RID α ; RID β and 14.7K.

3. A recombinant adenovirus according to claim 1 which is selected from any one
15 of SEQ ID NO:1, SEQ ID NO:2, SEQ ID NO:3, SEQ ID NO:4 or SEQ ID NO:13.

4. A recombinant adenovirus according to claim 1 which further comprises a
tissue specific promoter, a tumor specific promoter, or an inducible promoter substituted for
the E4 promoter.

5. A recombinant adenovirus according to claim 4 wherein the tissue-specific
20 promoter is a surfactant protein B promoter.

6. A recombinant adenovirus according to claim 5 which comprises SEQ ID
25 NO:14, SEQ ID NO:15 or SEQ ID NO:16.

7. A recombinant adenovirus according to claim 1, wherein the vector further
comprises a gene encoding an anti-cancer product.

8. A recombinant adenovirus according to claim 7, wherein the gene encoding an
30 anti-cancer product is in the E3 region of the vector.

9. A recombinant adenovirus according to any one of the preceding claims for
use in a method of treatment of a tumour in a patient.

10. Arecombinant adenovirus according to any one of the preceding claims
together with one or more replication-defective adenovirus which expresses an anti-cancer
gene product, for use in a method of treatment of a tumour in a patient, wherein the
recombinant adenovirus complements the spread of the replication-defective adenovirus in the
5 tumor.

11. Use of a recombinant adenovirus according to any one of claims 1 to 9 for the
manufacture of a medicament for the treatment of a tumour in a patient.

10 12. Use according to claim 11 wherein said tumour is further treated with
radiation.

13. Use according to claim 11 wherein said tumour is further treated with
chemotherapy.

15

14. A composition comprising the recombinant adenovirus of any one of claims 1
to 9, and a second recombinant virus which is replication defective and which expresses an
anti-cancer gene product, wherein the recombinant adenovirus complements replication of the
second recombinant virus.

20

15. A composition comprising the recombinant adenovirus of any one of claims 1
to 9, and a second recombinant virus which is replication competent in a neoplastic cell.

DATED THIS NINTH DAY OF JULY 2004

25 SAINT LOUIS UNIVERSITY

BY

PIZZEYS PATENT AND TRADE MARK ATTORNEYS

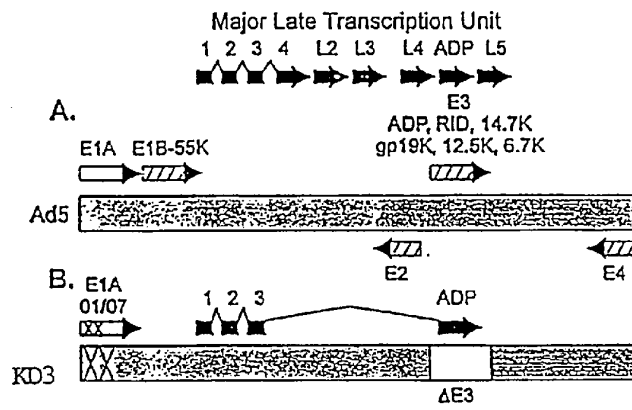


FIGURE 1

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ADP Is Expressed Earlier in Infection
By KD1, KD3, GZ1, and GZ3

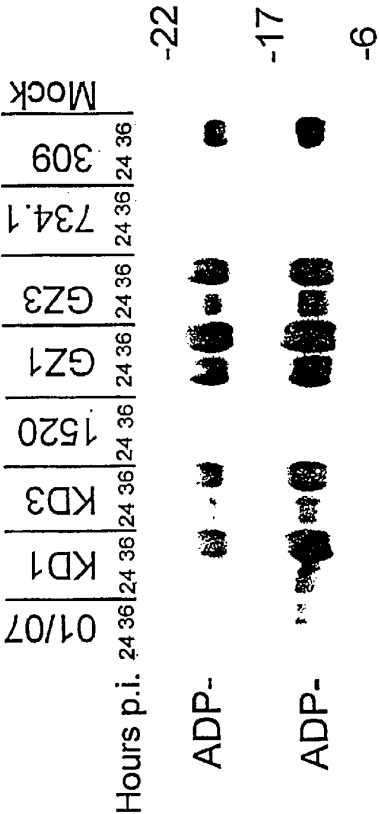


FIGURE 2

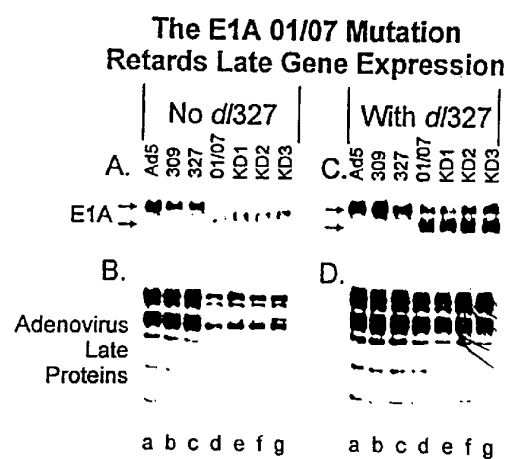


FIGURE 3

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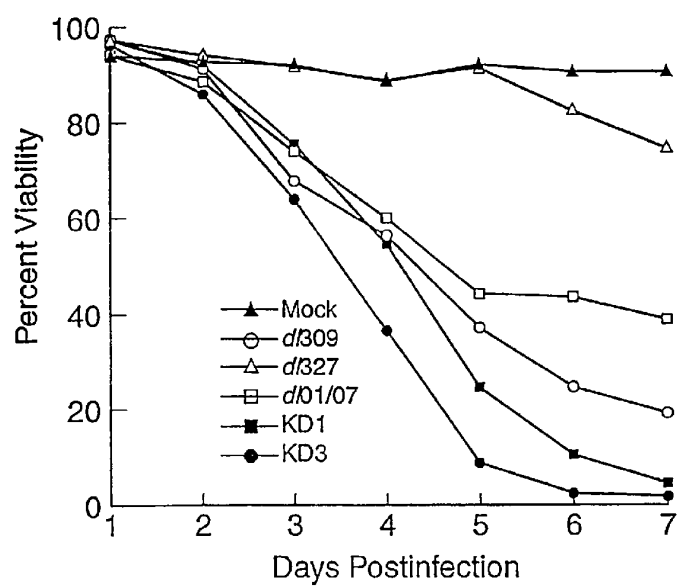


FIGURE 4

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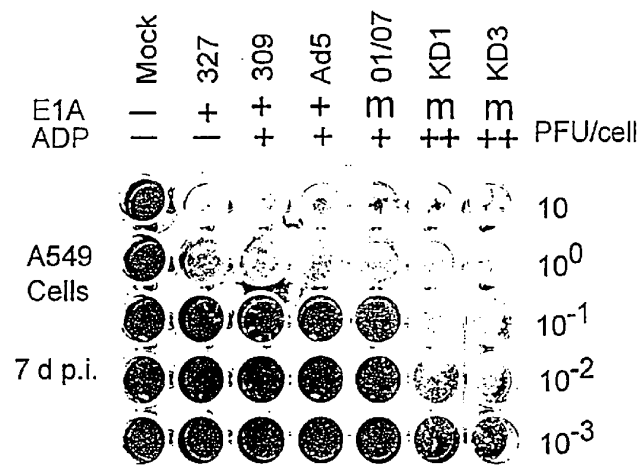


FIGURE 5

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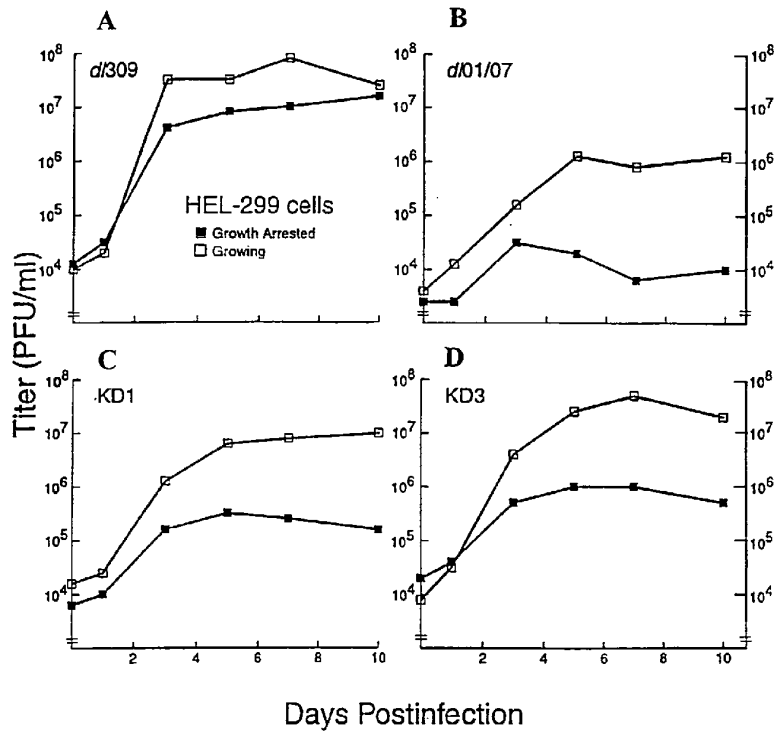


FIGURE 6

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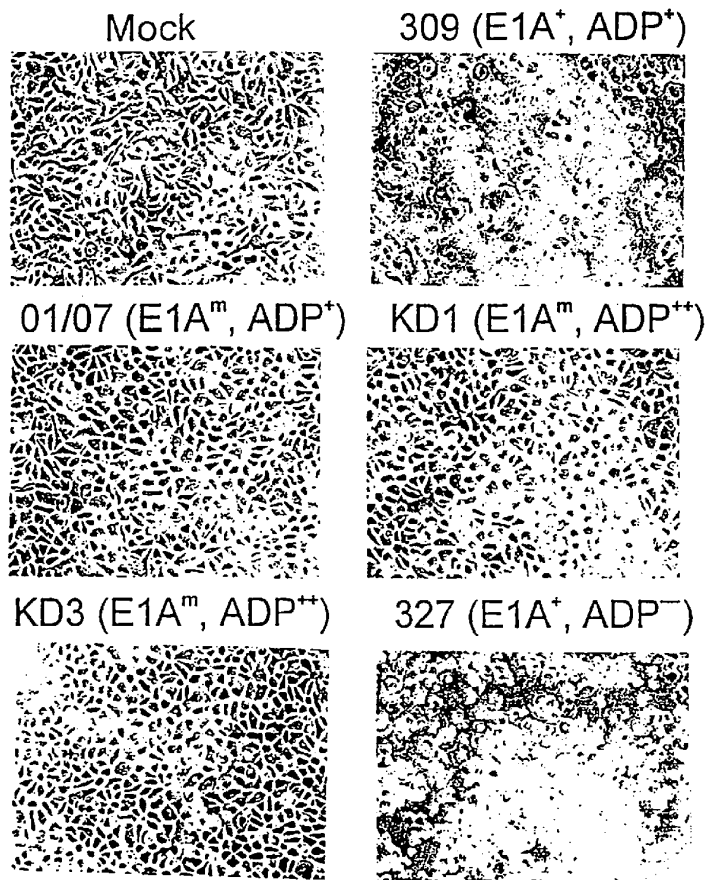


FIGURE 7

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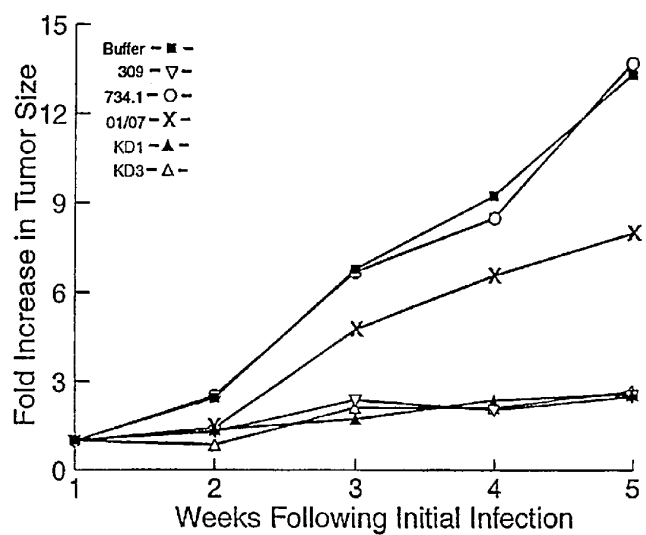
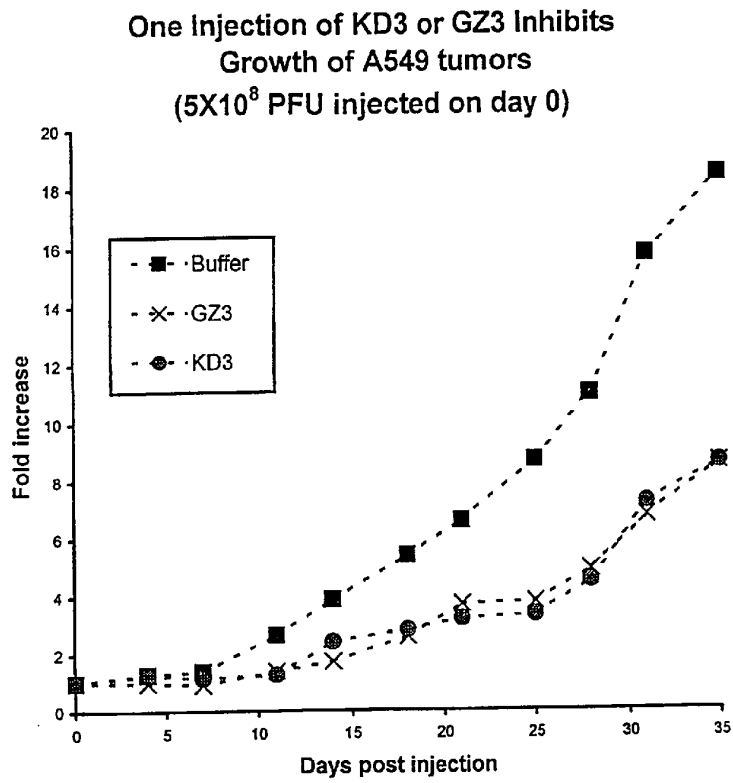


FIGURE 8A

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**FIGURE 8B**

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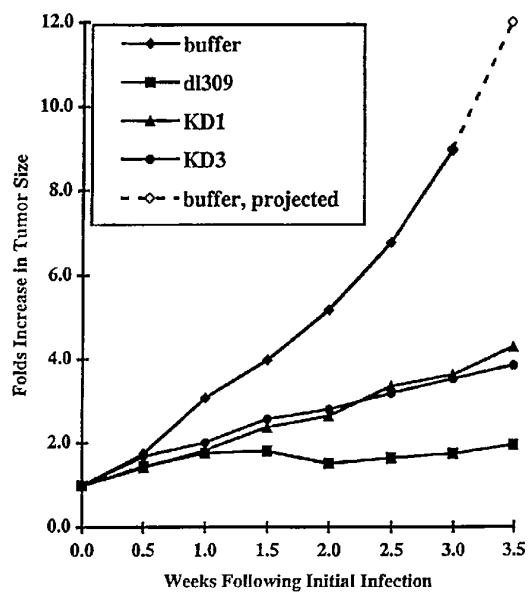


FIGURE 9

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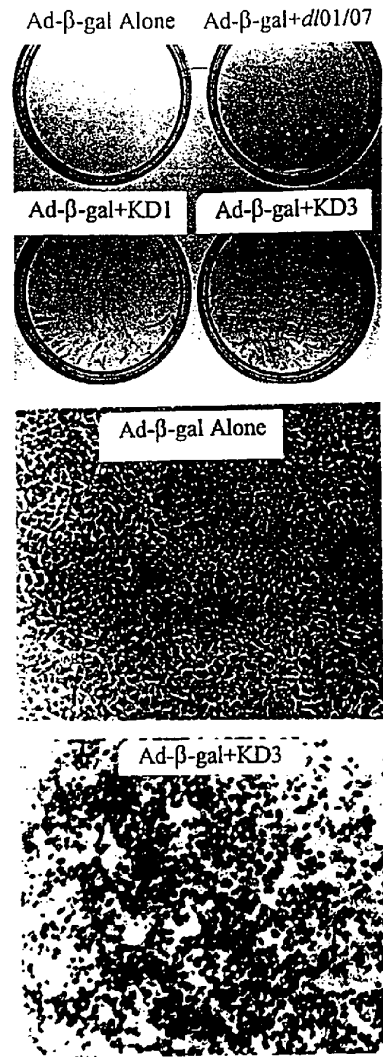


FIGURE 10

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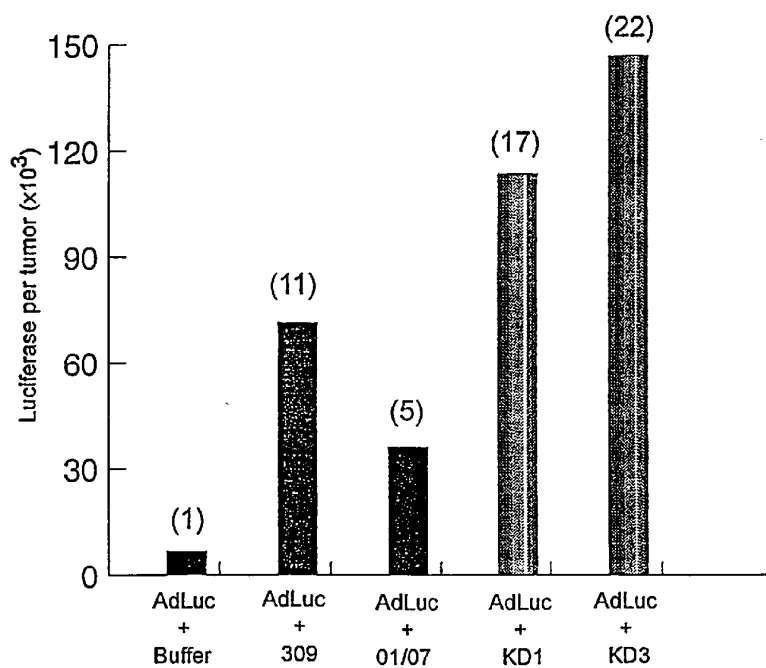


FIGURE 11

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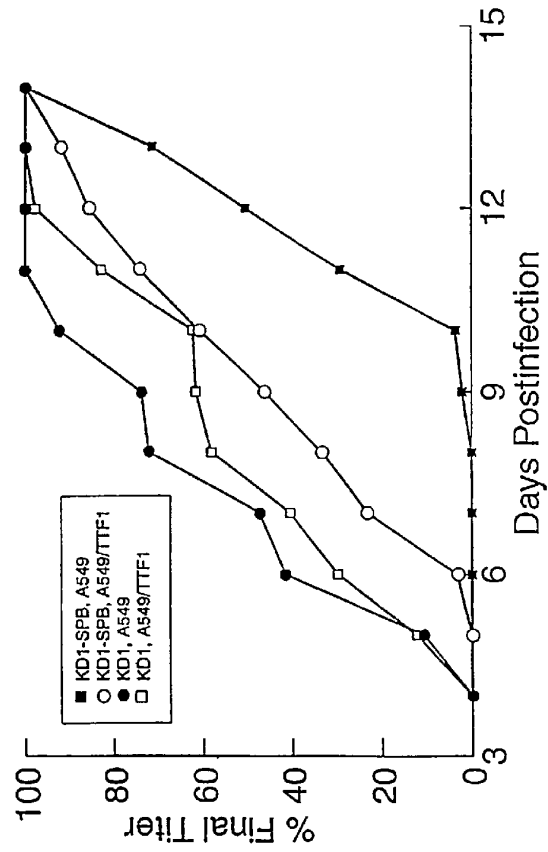


FIGURE 12

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**KD1-SPB With the SPB Promoter in Place of the E4
Promoter Grows on H44a Lung Cancer Cells with the
TTF1 Transcription Factor**

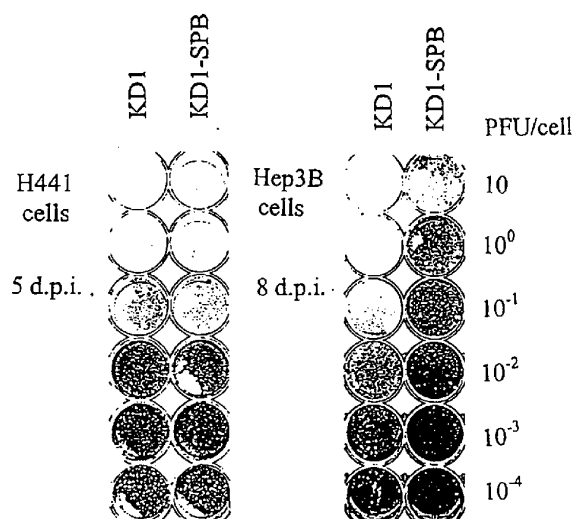


FIGURE 13

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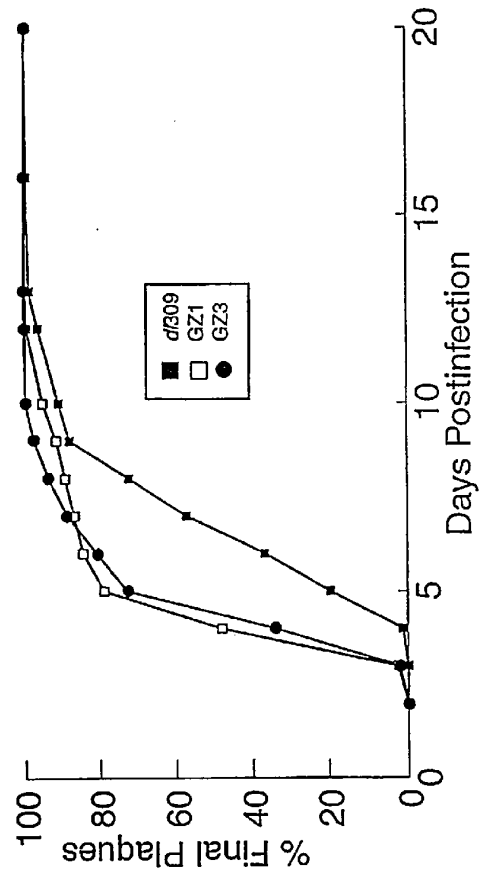


FIGURE 14

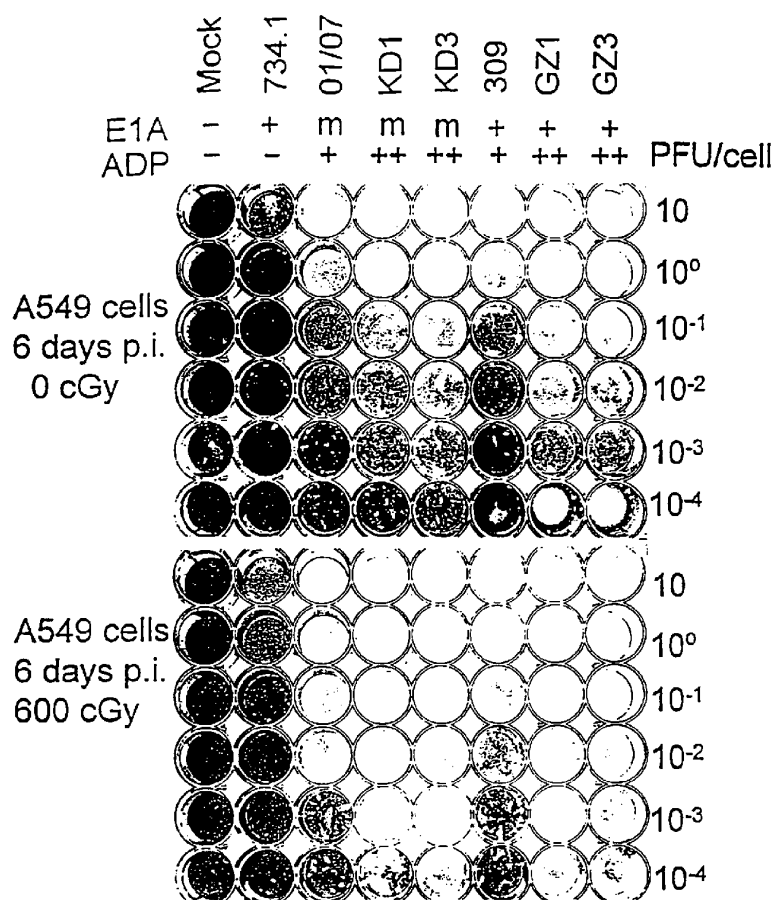


FIGURE 15

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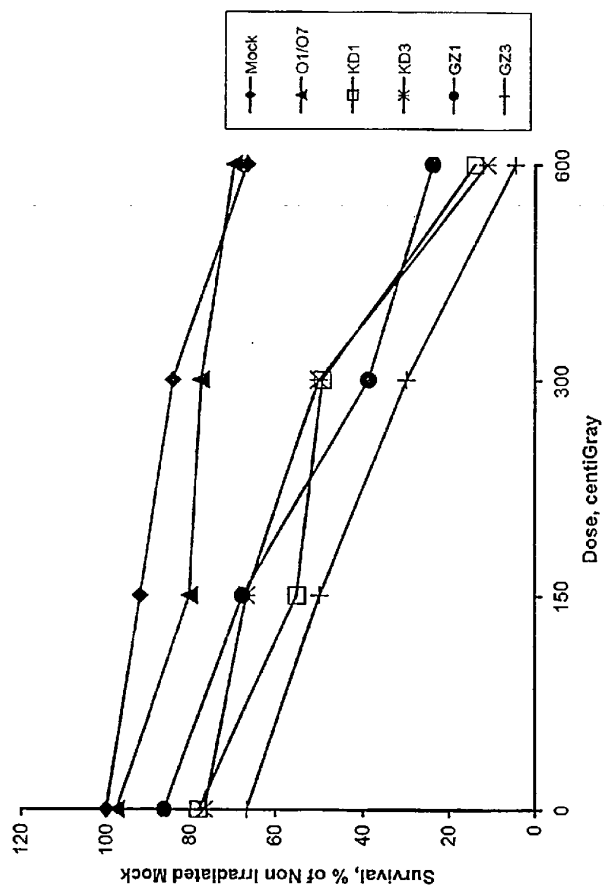


FIGURE 16

17166

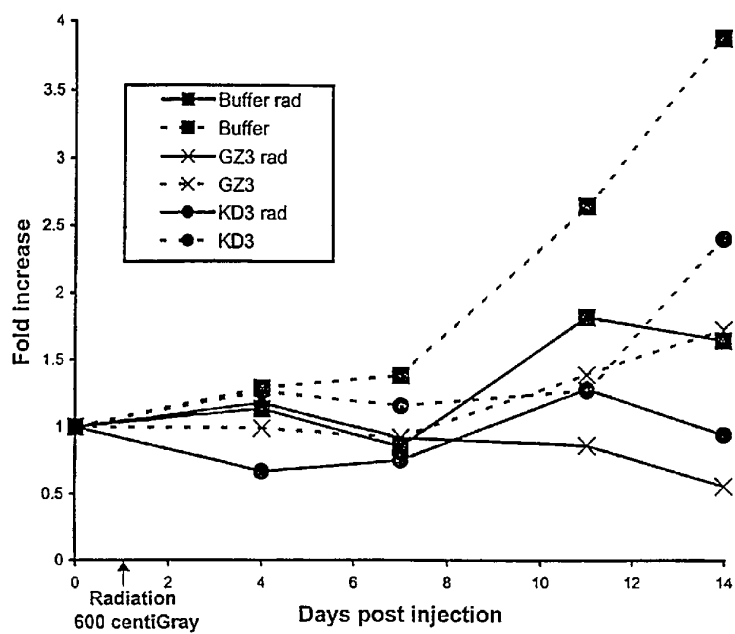


FIGURE 17

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Ad2 Adenovirus Death Protein*Luminal Domain*

MTGSTIAPTTTDYRNTTATGLTSALNLPQVHAFVND 35

O - glycosylation *N - glycosylation*

WASLDMWWFSIALMFVCLIMWLICCLKRRRARPP 70

Transmembrane
(Signal - Anchor)

Basic - Proline

IYRPIIVLNPHNEKIHRLDGLKPCSLLLQYD 101

Cytoplasmic - Nucleoplasmic Domain

FIGURE 18A

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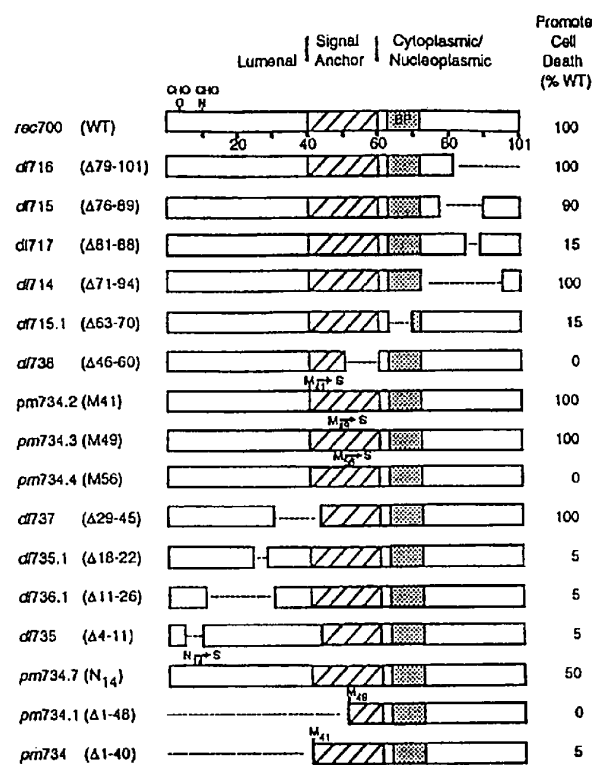


FIGURE 18B

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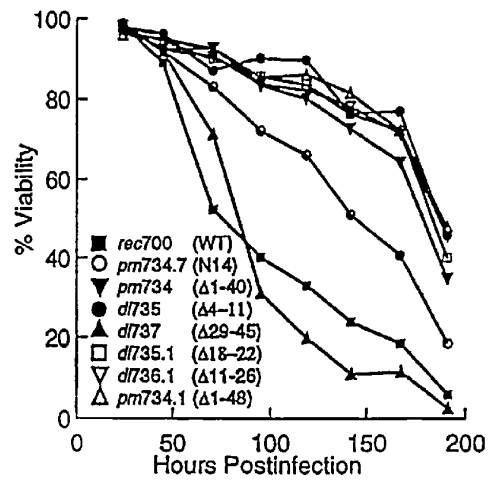


FIGURE 19A

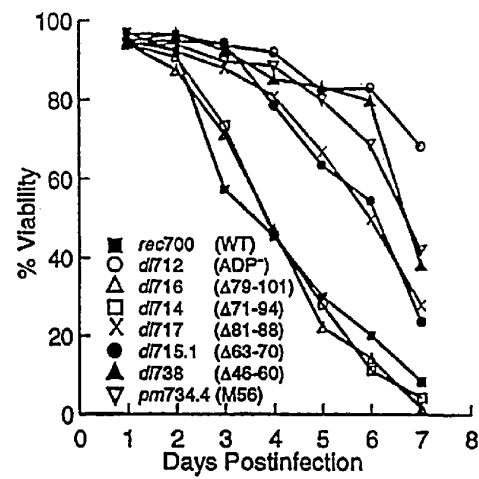


FIGURE 19B

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Seq ID No.	10	20	30	40	50
5 Ad1	-----MVD	T VNSYNTATGL	TSALNLPQVS	TFVNNWANLG	MWWFSIALMF
6 Ad2	MTGSTIAPT	T DYRNTTATGL	TSALNLPQVH	AFVNDWASLD	MWWFSIALMF
7 Ad5	-----MTN	T TNAAAATGL	TSTINTPQVS	AFVNNWDNLG	MWWFSIALMF
8 Ad6	-----MVD	T VNSYNTATGL	KSALNLPQVH	AFVNDWASLG	MWWFSIALMF
9 d1716	MTGSTIAPT	T DYRNTTATGL	TSALNLPQVH	AFVNDWASLD	MWWFSIALMF
10 d1715	MTGSTIAPT	T DYRNTTATGL	TSALNLPQVH	AFVNDWASLD	MWWFSIALMF
11 d1714	MTGSTIAPT	T DYRNTTATGL	TSALNLPQVH	AFVNDWASLD	MWWFSIALMF
12 d1737	MTGSTIAPT	T DYRNTTATGL	TSALNLPQ--	-----	-----IALMF

	60	70	80	90	100
5 Ad1	VCLIIIMWLS	C CLKRRRARPP	I YKPIIVLNP	NNDGIHRLDG	LNTCSFSFAV -
6 Ad2	VCLIIIMWLIC	C CLKRRRARPP	I YRPIIVLNP	HNEKIHRLDG	LKPCSLLQY D
7 Ad5	VCLIIIMWLIC	C CLKRRRARPP	I YSPIIVLHP	NNDGIHRLDG	LKHMFFSLTV -
8 Ad6	VCLIIIMWLIC	C CLKRRRARPP	I YRPIIVLNP	HNEKIHRLDG	LKPCSLLQY D
9 d1716	VCLIIIMWLIC	C CLKRRRARPP	I YRPIIVL--	-----	-----
10 d1715	VCLIIIMWLIC	C CLKRRRARPP	I YRPI-----	-----G	LKPCSLLQY D
11 d1714	VCLIIIMWLIC	C CLKRRRARPP	-----	-----	-----SLLQY D
12 d1737	VCLIIIMWLIC	C CLKRRRARPP	I YRPIIVLNP	HNEKIHRLDG	LKPCSLLQY D

Seq. ID No.

17	aa 1-40 of Ad2 ADP	MTGSTIAPT	T DYRNTTATGL	TSALNLPQVH	AFVNDWASLD
18	aa 41-59 of Ad2 ADP	MWWFSIALMF	VCLIIIMWLI		
19	aa 63-70 of Ad2 ADP	KRRRARPP			
20	aa 60-101 of Ad2 ADP	C CLKRRRARPP	I YRPIIVLNP	HNEKIHRLDG	LKPCSLLQY D

FIGURE 20

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LOCUS ad5 comple 35935 bp DNA SYN 06-FEB-1999
DEFINITION ad5 complete genome
ACCESSION ad5 comple
KEYWORDS
SOURCE Unknown.
ORGANISM Unknown
Unclassified.
REFERENCE 1 (bases 1 to 35935)
AUTHORS Self
JOURNAL Unpublished.
BASE COUNT 8367 a 10073 c 9761 g 7734 t
ORIGIN
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61 TTGTGACGTG GCGCGGGGCG TGGGAACGGG GCGGGTGACG TAGTAGTGTG GCGGAAGTGT
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361 GACTTTGACC GTTTACGTGG AGACTCGCCC AGGTGTTTTT CTCAGTGTTT TTCGCGGTTT
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1021 CAGGTCTTGT CATTATCACC GGAGGAATAC GCGGGACCCA GATATTATGT GTTCGCTTTG
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1261 CCAGAACCGG AGCCTGCAAG ACCTACCGCG CGTCTTAAA TGGCGCTGCG TATCTGAGA
1321 CGCCCGACAT CACCTGTGTC TAGAGAATGC AATAGTAGTA CGGATAGCTG TGACTCCGGT
1381 CCTTCTAACA CACCTCCTGA GATACACCCG GTGGTCCCGC TGTGCCCAT TAAACCAATT
1441 GCGGTGAGAG TTGGTGGGGG TCCCGAGGCT GTGGAATGTA TCGAGGACTT GCTTAACGAG
1501 CCTGGGCAAC CTTTGGACTT GAGCTGTAAA CCGCCAGGCG CATAGGTGT AAACCTGTGA
1561 TTGCGTGTGT GGTAAACGCC TTTGTTTGTG GAATGAGTTG ATGTAGTTT AATAAGGGT
1621 GAGATAATGT TTAACCTGCA TGGCGTGTGA AATGGGCGG GGCTTAAAGG GTATATAATG
1681 CGCCGTGGCG TAACTTGTGT TACATCTGAC CTCATGGAGG CTTGGGAGTG TTTGGAAGAT
1741 TTTTCTGCTG TCGTAACTT GCTGGAACAG AGCTCTAACA GTACCTCTTG GTTTGGAGG
1801 TTTCTGTGGG GCTCATCCCA GGCAAAGTTA GTCTGCAGAA TTAAGGAGGA TTACAAGTGG
1861 GAATTTGAAG AGCTTTTGAA ATCTGTGTGT GAGCTGTTTG ATTCTTTGAA TCTGGGTAC
1921 CAGGCGCTTT TCCAGAGAA GGTCAATCAG ACTTTGGATT TTTCCACACC GGGCGCGGCT
1981 GCGCGTGTGT TTGCTTTTTT GAGTTTTATA AAGGATAAAT GGAGCGAAGA AACCCATCTG
2041 AGCGGGGGGT ACCTGCTGGA TTTTCTGGCC ATGCATCTGT GGAGAGCGGT TGTGAGACAC
2101 AAGAATCGCC TGCTACTGTT GTCTTCCGTC CCGCCGCGCA TAATACCGAC GGAGGAGCAG
2161 CAGCAGCAGC AGGAGGAAGC CAGCGCGCGG CGGAGGAGC AGAGCCCATG GAACCCGAGA
2221 GCGCGCTTGG ACCCTCGGGA ATGAATGTTG TACAGGTGGC TGAACGTAT CCAAGAACTGA
2281 GAGCATTTT GACAAATTACA GAGGATGGGC AGGGGCTAAA GGGGGTAAAG AGGAGCGGG
2341 GGGCTTGTGA GGTACAGAG GAGGCTAGGA ATCTAGCTTT TAGCTTAATG ACCAGACACC
2401 GTCTCTGAGT TATTACTTTT CAACAGATCA AGGATAATTG CGCTAATGAG CTTGATCTGC
2461 TGGCGCAGAA GTATTCCATA GAGCAGCTGA CCACTTACTG GCTGCAGCCA GGGGATGATT
2521 TTGAGGAGGC TATTAGGGA TATGCAAGG TGGCACTTAG GCCAGATTGC AAGTACAAGA
2581 TCAGCAAACT TGTAAATATC AGGAATTGTT GCTACATTTT TGGGAACGGG GCGAGGTGG
2641 AGATAGATAC GGAGGATAGG GTGGCCTTTA GATGTAGCAT GATAAATATG TGGCCGGGGG

ad5

FIGURE 21
(SHEET 1)

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1

2701 TGCTTGGCAT GGACGGGGTG GTTATTATGA ATGTAAGGTT TACTGGCCCC AATTTIAGCG
2761 GTACGGTTTT CCTGGCCAAT ACCAACCTTA TCCTACACGG TGTAAGCTTC TATGGGTTTA
2821 ACAATACCTG TGTGGAAGCC TGGACCGATG TAAGGSGTTCG GGGCTGTGCC TTTTACTGCT
2881 GCTGGAAGGG GGTGGTGTGT CGCCCCAAAA GCAGGGCTTC AATTAGAGAA TGCTCTTTG
2941 AAAGGTGTAC CTGGGTATC CTGTCTGAAG GTAACCTCAG GGTGCGCCAC AATGTGGCCT
3001 CCGACTGTGG TTGCTTCATG CTAGTGAAAA GCCTGGCTGT GATTAGCAT AACATGGTAT
3061 GTGCAACTG CGAGGACAGG GCCTCTCAGA TGCTGACCTG CTCGGACGCG AACTGTCAAC
3121 TGCTGAAGAC CATTCACTA GCCAGCCACT CTCGCAAGGC CTGGCCAGTG TTTGAGCATA
3181 ACATACTGAC CGCTGTTC TTGCAITTTG GTAACAGGAG GGGGTGTTC CTACCTTACC
3241 AATGCAATTT GAGTCACACT AAGATATTGC TTGAGCCCGA GAGCATGTCC AAGGTGAACC
3301 TGAACGGGGT GTTTGACATG ACCATGAAGA TCTGGAAGGT GCTGAGGTAC GATGAGACCC
3361 GCACCAAGTG CAGACCCCTGC GAGTGTGGCG GTAAACATAT TAGGAACAGC CCGTGTATGC
3421 TGGATGTGAC CGAGGAGCTG AGGCCCGATC ACTTGGTGCT GGCCTGCACC CGCGCTGAGT
3481 TTGGCTCTAG CGATGAAGAT ACAGATTGAG GTACTGAAAT GTGTGGCGGT GGCCTAAGGG
3541 TGGGAAGAA TATATAAGGT GGGGCTCTTA TGTAGTTTGT TATCTGTTTT GCACGAGCCG
3601 CCGCCGCCAT GAGCACCAC TCCTTTGATG GAAGCAITGT GAGCTCATAT TTGACAACGC
3661 GCATGCCCC ATGGGCGGG GTGGTCTAGA ATGTGATGGG CTCCAGCATT GATGTGCGC
3721 CCGTCTGCG CGCAAACTCT ACTACCTTGA CCTACGAGAC CGTGTCTGGA ACGCCCTTGG
3781 AGACTGCAGC CTCGCGCGCC GCTTCAGCCG CTGCAGCCAC CGCTGTATCC GCCCGGATG
3841 ACTTTGCTTT CTTGAGCCCG CTTCAGAGCA GTGCAGCTTC CCGTGTATCC GCCCGGATG
3901 ACAAGTTTAC GGCTCTTTTG GCACAATTGG ATTCTTTGAC CCGGGAACCT AATGTGCTTT
3961 CTCAGCAGCT GTTGGATCTG CGCCAGCAGG TTTCTGCCCT GAAGGCTTCC TCCCTCCCA
4021 ATGCGGTTTA AAACATAAAT AAAAAACCAG ACTCTGTTTG GATTTGATC AAGCAAGTGT
4081 CTGTCTCTCT TTATTTAGGG GTTTTGCGCG CGCGGTAGGC CCGGACCCAG CGGTCTCGGT
4141 CGTTGAGGGT CCGTGTATTT TTTTCCAGGA CGTGGTAAAG GTGACTCTGG ATGTTGAGAT
4201 ACATGGGCAT AAGCCCGTCT CTGGGGTGA GGTAGCAGCA CTGCAGAGCT TCATGCTGCG
4261 GGGTGTGTGT GTAGATGATC CAGTCTGAGC AGGAGCGCTG GGCCTGGTGC CTAAGATGT
4321 CTTTCAGTAG CAAGCTGATT GCCAGGGGCA GGCCTTGGT GTAAGTGTTT ACAAGCGGT
4381 TAAGCTGGGA TGGGTGCATA CGTGGGATA TGAGATGCAT CTGTGACTGT ATTTTAAAGT
4441 TGGCTATGTT CCCAGCCATA TCCCTCCGGG GATTCATGTT GTGCAGAAC ACCAGCAGCAG
4501 TGTATCCGCT GCACCTGGGA AATTGTCTAT GTAGCTTAGA AGGAAATGCG TGGAAGAACT
4561 TGGAGACGCC CTTGTGACCT CCAAGATTTT CCATGCATTC GTCCATAATG ATGGCAATGG
4621 GCCCACGGGC GCGGCGCTGG GCGAAGATAT TTCTGGGATC ACTAACGTCA TAGTGTGTGT
4681 CCAGGATGAG ATCGTCATAG GCCATTTTAA CAAAGCGCGG GCGGAGGGTG CCAGACTGCG
4741 GTATAATGGT TCCATCCGGC CCAGGGCGGT AGTTACCTTC ACAGATTTCG ATTTCCACG
4801 CTTTGAGTTC AGATGGGGG ATCATGTCTA CCTGCGGGC GATGAAGAAA ACGGTTTCCG
4861 GGGTAGGGGA GATCAGCTGG GAAGAAAGCA GGTTCCTGAG CAGCTGCGAC TTACCGCAGC
4921 CCGTGGGCC GTAAATCACA CCTATTACCG GGTGCAACTG GTAGTTAAGA GAGCTGCAGC
4981 TGCGGTATC CCGTGCAGG GGGGCCACTT CGTTAAGCAT GTCCCTGACT CGCATGTTTT
5041 CCCTGACCAA ATCCGCCAGA AGGCGCTCGC CGCCAGCGA TAGCAGTTCT TGCAAGGAAG
5101 CAAAGTTTTT CAACGGTTTG AGACCGTCG CCGTAGGCAT GCTTTTGAGC GTTTGACCAA
5161 GCAGTTCAG GCGCTCCAC AGCTCGGTCA CCGTCTCTAC GGCATCTCGA TCCAGCATAT
5221 CTCCCTGTTT CCGCGGTTGG GCGGCTTTC GCTGTACGGC AGTAGTGGGT GCTGTGTCAG
5281 ACGGGCCAGG GTCATGTCTT TCCACGGGCG CAGGCTCTC GTCAGCGTAG TCTGGGTCCG
5341 GGTGAAGGG TCGCTCCGG GCTGCGCGCT GCGCAGGGTG CGCTGAGGCG TGACCATGGT
5401 GGTGCTAAG CGCTCGCGT CTTGCGCCTG CCGCTCGGCC AGGTAGCATT TGACCATGGT
5461 GTCATAGTCC AGCCCCCTCG CCGCGTGGCC CTTGGCGCGC AGCTTGCCCT TGAGGAGGCG
5521 GCGCACGAG GGGCAGTGCA GACTTTTGA GCGGTAGAGC TTGGGCGCGA GAAATACCGA
5581 TTCGGGGAG TAGGCATCCG CGCCGAGGCG CCGCAGAGC GTCTGCAATT CCACGAGCCA
5641 GGTGAGCTCT GCGCTTCGG GTTCAAAAC CAGGTTTCCC CCATGCTTTT TGATGCGTTT
5701 CTTACCTCTG GTTTCCATGA GCGGCTGTCC ACGCTCGGTG ACAGAAAGGC TGTCCGTATC
5761 CCGTATACA GACTTGAGAG GCCTGTCTCC GAGCGGTGTT CCGCGTCTCT CCGTATAG
5821 AAACCTCGAC CACTCTGAGA CAAAGGCTCG CCGTCCAGGC AGCAGCAAGG AGGCTAAGTG
5881 GAGGGGTAG CGGTCTGTGT CCACTAGGG GTCCACTCGC TCCAGGCTGT GAAGACACAT
5941 GTGCGCTCT TCGGCATCAA GGAAGGTGAT TGGTTGTAG GTGTAGGCCA CGTAGCCGGG
6001 TGTTCCTGAA GGGGGGCTAT AAAAGGGGT GGGGGCGCGT TCGTCTCAC TCTCTCCG
6061 ATGCTGTCT CCGAGGGCCA CCGTTGGGG TGAGTACTCC CTCTGAAAG CCGGCATGAC

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FIGURE 21
(SHEET 2)

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6121 TTCTGCGCTA AGATTGTCAG TTTCCAAAA CGAGGAGGAT TTGATATTCA CCTGGCCCGC
6181 GGTGATGCCT TTGAGGGTGG CCGCATCCAT CTGGTCAGAA AAGACAATCT TTTTGTGTTC
6241 AAGCTTTGGTG GCAAACGACC CGTAGAGGGC GTTGGACAGC AACTTTGGCGA TGGAGCGCAG
6301 GGTTTGGTTT TTGTCGGAT CGGCGGCTC CTGCGCGCG ATGTTTAGCT GCAAGTATTC
6361 GCGCGCAACG CACCGCCATT CGGAAAGAC GGTGGTGGC TCGTCGGSCA CCAGGTGCAC
6421 GCGCCAAACG CGGTTGTGCA GGTGACAAG GTCAACGCTG GTGGCTACCT CTCGCGTAG
6481 GCGCTCGTTG GTCCAGCAGA GCGGCGCGC CTGCGCGAG CAGAATGGCG GTAGGGGGTC
6541 TAGCTGCGTC TCGTCGGGG GGTCTGCGTC CACGGTAAAG ACCCGGGSCA GCAGGCGCGC
6601 GTCGAAGTAG TCTATCTTGC ATCCTTGCAA GTCTAGCGCC TGCTGCCATG CCGGGCGCGC
6661 AAGCGCGCGC TCGTATGGGT TGAGTGGGG ACCCATGGC ATGGGGTGGG TGAGCGCGGA
6721 GCGGTACATG CCGCAATGT CGTAAACGTA GAGGGGTCT CTGAGTATTC CAAGATATGT
6781 AAGGTAGCAT CTTCACCGC GGATGCTGGC GCGCACGTAA TCGTATAGTT CGTGGAGGG
6841 AGCGAGGAGG TCGGAGCCGA GGTGTCTACG GCGGGGTCT TCTGCTCGGA AGACTATCTG
6901 CCTGAAGATG GCATGTGAGT TGGATGATAT GGTGGACGC TGAAGACGT TGAAGCTGGC
6961 GTCTGTGAGA CCTACCGCGT CACGCAAGAA GGAGGCGTAG GAGTCGCGCA GCTTGTGTAC
7021 CAGCTCGCGC GTGACCTGCA CGTCTAGGGC GCAGTAGTCC AGGGTTTCTT TGATGATGTC
7081 ATACTTATCC TGTCCCTTTT TTTCCACAG CTGCGGTTG AGGACAACT CTCGCGGTTC
7141 TTTCCAGTAC TCTTGGATCG GAAACCGTC GCGCTCGAA CGGTAAAGGC CTAGCATGTA
7201 GAACTGGTTG ACGCGCTGGT AGGCGCAGCA TCCCTTTTCT ACGGGTAGCG CGTATGCGTC
7261 CCGCGCCTTC CCGAGCGAGG TGTGGTGGAG CGCAAGGGT TCCTGACCA TGACTTTGAG
7321 GTACTGTGAT TTGAAGTCAG TGTGTCGCA TCCGCGTGC TCCAGAGCA AAAAGTCCGT
7381 CGCTCTTTTG GAACGCGGAT TTGGCAGGGC GAAGGTGACA TCGTTGAAGA GTACTTTCC
7441 CCGCGCGAGC ATAAAGTTGC GTGTGATGCG GAAGGGTCCC GGCACCTCGG AACGGTGTG
7501 AATTACCTGG GCGCGCAGCA CSATCTGCTC AAAGCGGTG ATGTGTGGC CCACAATGTA
7561 AAGTTCGAAG AAGCGCGGGA TGCCCTTGAT GGAAGGCAAT TTTTAAAGTT CCGTAGGT
7621 GAGCTCTTCA GGGGAGCTGA GCGCGTCTC TGAAGGGCC CAGTCTGCAA GATGAGGGT
7681 GGAAGCGACG AATGAGCTCC ACAGGTACAG GGCATTAGC ATTGCGAGT GGTCCGCAAA
7741 GGTCTCTAAG TGGCGACCTA TGGCCATTTT TTCTGGGGTG ATGCGTAGA AGGTAAAGCG
7801 GTCTGTGTCC CAGCGGTCCC ATCCAGGTTT CCGGCTAGG TCTCGCGCG CAGTCACTAG
7861 AGGCTCATCT CCGCGAACT TCAAGACAG CATGAAGGGC ACGAGCTGCT TCCCAAGGC
7921 CCCATCCAA GTATAGTCT CTACATCGTA GGTGACAAAG AGACGCTCGG TCGCAGGATG
7981 CGAGCGATC GGAAGAACT GGATCTCCCG CCACCAATG GAGGAGTGGC TATTGATGT
8041 GTGAAAGTAG AAGTCCCTGC GACGCGCGGA ACATCTGTG TGGCTTTGT AAAAACCGTC
8101 GCACTACTGG CAGCGGTGCA CCGGCTGTAC ATCTGACAG AGGTTGACCT GACGACCGCG
8161 CACAAGGAAG CAGAGTGGGA ATTTAGGCC CTGCGCTGGC GGGTTTGGCT GGTGTCTTC
8221 TACTTGGCT GCTGTCTCT GACCGTCTGG CTGCTCGAG GAGGTTACGG TGGATCGGAC
8281 CACCACGCGC CGGAGCCCA AAGTCCAGAT GTCCGCGCG GCGGCTCGCA GGTGAGCGG
8341 AACATCGCGC AGATGGGAGC TGTCCATGTT CTGAGCTCC CCGGCTAGAT CCAGGTGATA
8401 GAGCTCTGTC AGGTTTACCT CGCATAGAGC GTGATGGCT TGCAAGAGGC CGCATCCCG
8461 CCTAATTTC AGGGCTGGT TGGTGGCGGC GTGGGCGCG GGGGTGTCCT TGGATGATG
8521 CCGCGGACT ACGGTACCGC GCGGCGGGCG GTGGGCGCG GGGGCTCCGG ACCCGCGGG
8581 ATCTAAAGC GGTGACGCG GCGAGCCCC GGAGGTAGG GGGGCTCCGG ACCCGCGGG
8641 AGAGGGGCGA GGGGACGTC GCGCGCGCG CCGGGCAGGA GCTGGTCTG CCGCGTAGG
8701 TTGCTGCGCA ACGGACGAC GCGCGGTTG ATCTCTGAA TCTGGCGCT CTGCGTGAAG
8761 ACGAGGGGCC CGGTGAGCTT GAGCCTGAAA GAGAGTTCGA CAGAATCAAT TCGGTGTGCG
8821 TTGACGCGCG CCGGCGCAA AATCTCTGTC ACCTCTCTG AGTTGTCTTG ATAGGCGATC
8881 TCGGCCATGA ACTGCTGAT CTCTTCTCT TGSAGATCT CCGCTCCGCG TCGCTCCAGC
8941 GTGGCGCGCA GGTCTGTGGA AATCGGGCG ATGAGCTGCG AGAAGCGGT GAGGCTCCCG
9001 TCGTTCAGA CGCGCTGTA GACCAAGCCC CTTGCGCAT CCGGGCGCG CATGACCAAC
9061 TGCGCGAGAT TGAGCTCCAC GTGCGGGCG AAGACGCGT AGTTTCGAG GCGCTGAAAG
9121 AGGTAGTTGA GGTGGTGGC GGTGTGTTCT GCCACGAAGA AGTACATAAC CCAGCGTCG
9181 AACGTGATT CGTTGATATC CCGAAGGCT TCAAGGCGCT CCATGGCTCT GTAGAGTCC
9241 ACGCGAAGT TGAAGAACTG GAGTTGCGC GCGACACGG TTAATCTCT CTCAGAGGA
9301 CGGATGAGCT CCGGACAGT GTGCGCACG TCGCGCTCAA AGCTACAGG GCGCTCTCT
9361 TCTTCTTCAA TCTCTCTTC CATAAGGCG TCCCTTCTT CTCTCTCTG CCGCGTGGG
9421 GGAGGGGGA CACGCGGCG ACGACGCGC ACCGGGAGC GGTGACAAA GCGCTCGATC
9481 ATCTCCCGC GCGACGCGC CATGCTCTG GTGACGCGC GCGCTTCTC CCGGGGCGC

FIGURE 21
(SHEET 3)

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9541 AGTTGGAAGA CGCGGCCGT CATGTCCCGG TTATGGGTTG GCGGGGGGCT GCCATGCGGC
9601 AGGGATACGG CGCTAACGAT GCATCTCAAC AATTGTTGTG TAGGTACTCC GCCGCGAGG
9661 GACCTGAGCG AGTCCGCATC GACCGGATCG GAAAACCTCT CGAGAAAGGC GTCTAACCCAG
9721 TCACAGTCGC AAGGTAGGCT GAGCACCCTG CCGGCGGCA CCGGCGGCG GTCCGGGTTG
9781 TTTCTGGCGG AGGTGCTGCT GATGATGTAA TTAAGTAGG CGTCTTGAG ACGGCGGATG
9841 GTCCAGAGAA GCACCATGTC CTGGGTCGG GCCTGTGAA TGCGCAGCG GTCCGCCATG
9901 CCCGAGCTT CGTTTGGACA TCGGCGCAGG TCTTTGTAGT AGTCTTGAT GAGCCTTCT
9961 ACCGCACTT CTTCTTCTCC TTCTCTTGT CTTCATCTC TTGCATCTAT CGCTGCGGG
10021 GCGGCGGAGT TTGGCGGTAG GTGGCGCCCT CTCTCTCCA TGCGTGTGAC CCGAAGCCC
10081 CTCATCGGCT TAGGACAGGC TAGGTGCGCG ACAACGCGCT CGGTAAATAT GGCTGTCTG
10141 ACCTCGTGTA GGTAGACTG GAAGTCAATC ATGTCCACAA AGCTCTGGTG ACCCGCTG
10201 TTGATGGTGT AAGTGAGATT GGCCATAACG GACCACTTAA CGGTCTGGTG ACCCGCTG
10261 GAGAGCTCGG TGTACCTGAG ACGCGAGTAA GCCCTCGAGT CAAATACGTA GTCTTGCA
10321 GTCCGACCCA GGTACTGGTA TCCACCAAAA AAGTGGCGCG CCGGCTGGCG GTAGAGGGC
10381 CAGCGTAGGG TGGCGCGGG TCCGCGGGCG AGATCTTCCA ACATAAGCG ATGATATCCG
10441 TAGATGTACC TGGACATCCA GGTGATGCTT GCGCAGCGCG AAAAGTGTCT CCATGTCGG GACGCTCTG
10501 CGGACGCGGT TCCAGATGTT GGTGAGCGCT TAGACCGTGC CCAAGGAGAG CCGTGAAGC
10561 CCGGTGAGGC GCGCGCAATC GTTGCAGCTC TGGATAAATT CGCAGGGTAA TCATGGCGGA CAGCGCGGT
10621 GGCACCTCTT CCGTGGTCTG TGGATAAATT CGCAGGGTAA TCATGGCGGA CAGCGCGGT
10681 TCGAGCGCGG TATCCGCGCG TCCGCGCGTA TCCATGCGGT TACCGCGCG GTGTCGAAC
10741 CAGGTGTGCG ACCTCAGACA ACGGCGGAGT GCTCCTTTTG GCTTCTTCC AGGCGCGCG
10801 GCTGCTGCGG TAGCTTTTTT GGCCATGCG CGCGCGCAGC GTAAAGCGGT AGGCTGGA
10861 GCGAAGCAT TAAGTGGCTC GCTCCTGTGA GCGGAGGGT TATTTTCAA GGTGTGATC
10921 GCGGACCCCG CGGTTCGAGT CTCGACCGCG CCGGAGTGC GCGACGAGCC CTTTTTTTG
10981 CCGTCATGCA AGACCCCGCT TGCATAATCC TCCGAAACA GCGACGAGCC CTTTTTTTG
11041 TTTTCCCAAG TGCATCCGCT GTGCGCGCAG ATGCGCCCCC CTCCTCAGCA GCGCGAGAG
11101 CAAAGACAGA GGCAGACATG CAGGCGACCC TCCCTCTCTC CTACCGCGTC AGGAGGGCG
11161 ACATCCGCGG TTGACGCGG AGCAGATGCT GATTACGAAC CCGCGCGCG CCGGCGCGG
11221 CACTACCTGG ACTTGGAGGA GGGCGAGGCG CTGGCGCGCG TAGGAGCGCC CTCCTCTGAG
11281 CGGTACCCAA GGTGCGAGCT GAAGCGTGAT ACGGTGAGG CGTACGTGCC GCGCGAGAG
11341 CTGTTTCGCG ACCGCGAGGG AGAGGAGCCC GAGGAGATGC GGGATCGAAA GTTCCACGCA
11401 GCGCGCGAGC TGGCGCATG CCTGAATCGG GAGCGGTTGC TCGCGGAGGA GGCATTTGAG
11461 CCGGACGCGC GAACCGGGAT TAGTCCCGCG CGCGCACACG TGGCGGCCCG CGACCTGCTA
11521 ACCGCTACG AGCAGACGGT GAACCGAGG ATTAACCTTC AAAAAAGCTT TAACRACCA
11581 GTGCTGACG TTGTGCGCG CGAGGAGGTG GCTATAGGAC TGATGCATCT GTGGGACTTT
11641 GTAGCGCGC TGGAGCAAAA CCCAAATAGC AAGCGCTCA TGGCGCAGCT GTTCTTTATA
11701 GTGACGACA CGAGGGACAA CGAGGCAATC AGGATGCGC TGCTAAACAT AGTAGAGCC
11761 GAGGCGCGCT GGCTGCTCGA TTTGATAAAC ATCTGCGAGA GCATAGTGTG GCAGGAGCG
11821 AGCTTGAGCC TGGCTGACAA GGTGGCGCGC ATCAACTATT CCATGCTTAG CCGGCGAAG
11881 TTTTACGCCC GCAAGATATA CCATACCCCT TACGTTCCCA TAGACRAGGA GGTAAAGATC
11941 GAGGGTTCT ACATGCGCAT GCGCTGAAG GTGCTTACCT TGAGCGACGA CCGTGGCGTT
12001 TATCGCAACG AGCGCATCCA CAAGGCGGTG AGCGTGAGCC GCGCGCGCG GCTCAGCGAC
12061 CGCGAGCTGA TGCACAGCCT GCAAGGGGCC CTGGCTGGCA CCGGCGAGCG CGATAGAGAG
12121 GCGGAGTCTT ACTTTGACCG GCGGCTGAC CTGCGCTGGG CCGCAAGCGC ACGCGCCCTG
12181 GAGGCGAGCT GGGCGGACCG TGGGCTGGCG GTGGCACCGG CCGCGCTGG CACGCTGGC
12241 GCGCTGAGG AATATGACGA GACGATGAG TACGAGCCAG AGGACGCGCA GTACTAAGCG
12301 TGATGTTTC TGATCAGATG ATGCAAGACG CAACGAGCCC GCGGCTGCGG GCGCGCTG
12361 AGAGCCAGCC GTCCGCGCTT AACTCCACCG ACGACTGCGG CCGGCTCATG GACCGCATCA
12421 TGTGCTGAC TGCAGCAAT CCTGACGCGT TCCGCGACGA CCGCGAGGCC AACCGCTCT
12481 CCGCAATTCT GGAAGCGGTG GTCCGCGCG CCGCAAAACC CACGCAAGAG AAGGTGCTG
12541 CGATCGTAAA CCGCTGCGC GATTCAGCGC GTGGCTGCTT ACAACAGCG CAACTGACG ACCAACCTG
12601 ACGACGCGCT GCTTCAGCGC GCGGAGCGCG TGGCGCAGCG TGAGCGCGCG CAGCAGCAGG
12661 ACGGCTGCT GGGGATGTT GCACTAAACG CTTCTCTGAG TACACAGCCC GCCAAGTGC
12721 GCAACCTGGG CTCCATGCTT ACCAATTTT TGAGCGCACT GCGGCTAATG GTGACTGAGA
12781 CCGCGGACGA GAGGAGTAC ACCAATTTT TGAGCGCACT GCGGCTAATG GTGACTGAGA
12841 CACCGCAAG TGAGGTGTAC CAGTCTGGG CAGACTATTT TTTCCAGACC AGTAGACAAG
12901 GCCTGCAGAC CGTAAACCTG AGCCAGGCTT TCAAAAACCTT GCGAGGCGTG TGGGGGTG

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FIGURE 21
(SHEET 4)

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12961 GGGCTCCAC AGGCGACGCG GCGACGCTGT CTAGCTTGCT GACGCCAAC TCGCGCTGT
13021 TGCTGTGCT AATAGCGCCC TTCACGGACA GTGGCAGGCT GTCCGGGAC ACATACCTAG
13081 GTCACCTGCT GACACTGTAC CCGGAGGCCA TAGGTCAGGC GCATGTGGAC GAGCATACTT
13141 TCCAGGAGAT TACAAGTGTC AGCCGCGCGC TGGGGCAGGA GGACACGGGC AGCCTGGAGG
13201 CAACCCATAA CTACCTGCTG ACCAACCGGC GGCAGAAAGT CCCTCTGTTG CACAGTTTAA
13261 ACAGCGAGGA GGAGCGCATT TTGCGCTACG TGCAGCAGAG CGTGAGCCTT AACCTGATGC
13321 GCGACGGGGT AACGCCCAGC GTGGCGCTGG ACATGACCGC GCGCAACATG GAACCGGGCA
13381 TGTATGCTTC AAACCGGCGG TTTATCAACC GCCTAATGGA CTACTTGATG CCGCGGGCGG
13441 CCGTGAAACC CGAGTATTTT ACCAATGCCA TCTTGAACCC GCACCTGGCTA CCGCCCCCTG
13501 GTTTCTACAC CGGGGGATTG GAGGTGCCCC AGGTAACGA TGGATTCTCT TGGACGACA
13561 TAGACGACAG CGTGTTTTCC CCGCAACCGC AGACCTTGCT AGAGTTCGAA CAGCGGAGC
13621 AGGCAGAGGC GCGCTGCGA AAGGAAAGCT TCCGAGGCC AAGCAGCTTG TCGATCTAG
13681 GCGCTGCGCG CCGCGGTCG GATGCTAGTA GCCCATTTCC AAGCTTGATA GGGTCTCTTA
13741 CCAGCACTCG CACCAACCGC CCGCGCTGCG TGGGCGAGGA GGAGTACCTA AACCACTGCG
13801 TGCTGACGCG GCAGCGCGAA AAAAACCCTG CTCGCGCATT TCCCAACAC GGGATAGAGA
13861 GCCTAGTGGA CAAGATGAGT AGATGGAAGA CGTACGCGCA GGAGCACAGG GACGTGCCAG
13921 GCCCGCGCCC GCCACCCGCT CGTCAAGGCG ACGACCGTCA GCGGGGCTG GTGTGGAGG
13981 ACGATGACTC GGCAGACGAC AGCAGCGTCC TGGATTGCGG AGGAGTGGC AACCCGTTG
14041 CGCACTTCG CCCCAGGCTG GGGAGAATGT TTTAAAAAAA AAAAAGCATG ATGCAAAATA
14101 AAAAATCTAC CAAGGCCATG GCACCGAGCG TTGGTTTCTT TGTATTCCCC TTAGTATGCG
14161 GCGCGCGCGC ATGTATGAGG AAGGTCTTCC TCCTCTCTAC GAGAGTGTGG TAGCGCGCGC
14221 GCCAGTGGCG GCGCGCTGG GTTCTCCCTT CGATGCTCCC CTGGACCGCG CGTTGTGCC
14281 TCCGCGTAC CTGCGGCTTA CCGGGGGGAG AAACAGCATC CGTTACTCTG AGTTGGCACC
14341 CCTATTGCG ACCACCCGCT TGTACTGTGT GGACAACAAG TCAACGGATG TGGCATCCCT
14401 GAACATACAG AACGACCAAC GCAACTTTCT GACCACGGTC ATTTAAAAA ATGACTACAG
14461 CCGCGGGGAG GCAAGCACAC AGACCATCAA TCTTGACGAC CGGTGCGACT GGGCGCGCA
14521 CCTGAATAAC ATCTTGCTA CCAACATGCC AAATGTGAAC GAGTTCATGT TTCCAATAA
14581 GTTTAAGCG CGGTGATGG TGTGCGCTT GCCTACTAAG GACAATCAGG TGGAGCTGAA
14641 ATACGAGTGG GTGGAGTTCA CGCTGCCCGA GGGCACTAC TCCGAGACCA TGACCATAGA
14701 CCTTATGAAC AACCGGATCG TGGAGCACTA CTTGAAAGTG GGCAGACAGA ACGGGTCTT
14761 GGAAAGCGAC ATCGGGGTAA AGTTTGACAC CGGCAACTTC AGACTGGGGT TTGACCCCGT
14821 CACTGGTCTT GTCATGCGCT GGGTATATAC AAACGAAGCC TTCCATCCAG ACATCATTTT
14881 GCTGCCAGGA TGCGGGGTGG ACTTCACCCA CAGCCGCTG AGCAACTTGT TGGCATCCG
14941 CAAGCGGCAA CCCTTCCAGG AGGGCTTTAG GATCACCTAC GATGATCTGG AGGGTGTAA
15001 CATTCGCCCA CTGTTGGATG TGGACGCTTA CCAGGCGAGC TCGGCGGAAG AGAACTCCA
15061 GGGCGGGGGT GCGCGAGGCG GCAGCAACAG CAGTGGCAGC GCGCGGGAAG AGAACTCCA
15121 CCGCGCAGCC GCGCGCATGC AGCCGGTGGG GGACATGAAC GATCATGCCA TTCCGCGCA
15181 CACCTTTGCC ACACGGGCTG AGGAGAAGCG CGCTGAGGCC GAAGCAGCGG CCGAAGCTGC
15241 CGCCCCCGCT GCGCAACCCG AGGTGAGGAA GCCTCAGAAG AAACCGGTGA TCAACCCCT
15301 GACAGAGGAC AGCAAGAAAC GCAGTTACAA CCTAATAAGC AATGACAGCA CCTTACCCA
15361 GTACCGCAGC TGTACCTTG CATACAACTA CCGCGACCTC CAGACCGGAA TCCGCTCATG
15421 GACCTCTCTT TGCACCTCTG ACGTAACCTG CGGCTCGGAG CAGGTCTACT GGTCTGTGCC
15481 AGACATGATG CAAGACCCCG TGACCTTCCG CTCCACGCGC CAGATCAGCA ACTTCCGGT
15541 GGTGGGCGCC GAGCTGTGCT CCGTGCACTC CAAGAGCTTC TACAACGACC AGGCGTCTA
15601 CTCCCAACTC ATCCGCCAGT TTACCTCTCT GACCCACGTG TTCAATCGCT TTCCGAGAA
15661 CCAGATTTTG GCGCGCCGCG CAGCCCCCAC CATCACACC GTCAGTGAAA AGTTCTCTG
15721 TCTACAGAT CACGGGAGCG TACCGCTGCG CAACAGCATC GAGGAGTCC AGCGAGTGAC
15781 CATTACTGAC GCCAGAGCGC GCACCTGCCC TACGTTTAC AAGGCCCTGG GCATAGTCTC
15841 GCGCGCGCTC CTATCGAGCC GCACCTTTTG AGCAAGCATG TCCATCTTTA TATCGCCAG
15901 CAATAACACA GGCTGGGGCC TGCGCTTCCC AAGCAAGATG TTTGGCGGG CCAAGAGCG
15961 CTCGACCAA CACCCAGTGC GCGTGCGCGG GCATACCGC GCGCCCTGGG GCGCGACAA
16021 ACGCGGCGC ACTGGGCGCA CCACCGTGA TGACGCCATC GACCGGCTG TGGAGGAGC
16081 GCGCACTAC ACGCCACGCG CGCCACCACT GTCCACAGTG GACCGGCCA TTCAGACCGT
16141 GGTGCGCGGA GCGCGCGCT ATGCTAAAT GAAGAGACGG GCGAGGCGCG TAGCACGTG
16201 CCACGCGCGC GACCGCGCGC CTGCGGCCA ACGCGCGCG GCGCGCTGCT TTAACCGCGC
16261 ACGTGCACCC GCGCGACGG GCGCATGCG GCGCGCTCGA AGGCTGGCGG CCGGTATTGT
16321 CACTGTGCC CCCAGGTCCA GCGCAGAGC GCGCGCGCA GCACCGCGG CCATTAGTGC

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FIGURE 21
(SHEET 5)

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16381 TATGACTCAG GGTGCGAGGG GCAACGTGTA TTGGGTGCGC GACTCGGTTA GCGGCCTGCG
 16441 CGTGCCCGTG CGCACCCGCG CCCGCGCGCA CTAGATTGCA AGAAAAAAT ACTTAGACTC
 16501 GTACTGTTGT ATGTATCCAG CGCGCGCGGC GCGCAACGAA GCTATGTCCA AGCGCAAAAT
 16561 CAAAGAAGAG ATGCTCCAGG TCATGCGGCC GGAGATCTAT GSCCCCCCGA AGAAGGAAGA
 16621 GCAGGATTAC AAGCCCGAA AGCTAAAGCG GGTCAAAAAG AAAAAGAAAG ATGATGATGA
 16681 TGAACCTGAC GACGAGGTGG AACTGCTGCA CGCTACCGCG CCCAGGCGAC GGGTACAGTG
 16741 GAAAGTTGCA CGCGTAAAC GTGTTTGGG ACCCGGCACC ACCGTAGTCT TTACGCCCGG
 16801 TGAGCGCTCC ACCCGCACCT ACAAGCGCGT GTATGATGAG GTGTACGGCG ACGAGGACCT
 16861 GCTTGAGCAG GCCAACGAGC GCCTCGGGGA GTTTGCCCTAC GGAAAGCGGC ATAAGGACAT
 16921 GCTGGCGTTG CCGCTGACG AGGCGAACCC AACACCTAGC CTAAAGCCCG TAACACTGCA
 16981 GCAGGTGCTG CCGCGCGTTG CACCGTCCGA AGAAAAGCGC GGCCTAAAGC CGGAGTCTGG
 17041 TGACTTGGCA CCCACCGTGC AGCTGATGAT ACCCAAGCGC CAGCGACTGG AAGATGTCTT
 17101 GGAAAAAATG ACCGTGGAAC CTGGGCTGGA GCCCGAGGTC CGCGTGCGGC CAATCAAGCA
 17161 GGTGCGCGCG GGAAGTGGCG TGCAGACCGT GGACGTTGAG ATACCCACTA CCAGTAGCAC
 17221 CAGTATTGCG ACCCGCACAG AGGGCATGGA GACACAAACG TCCCGGTTG CCTCAGCGGT
 17281 GCGGATGCGC GCGGTGCGAG CGGTGCTGCG GCGCGGTCC AAGACCTCTA CGGAGGTGCA
 17341 AACGCAACCG TGGATGTTTC GCGTTTCAGC CCGCGCGCGC CCGCGCGGTT CGAGGAAGTA
 17401 CCGCGCGCGC AGCGCGCTAC TGCCCGAATA TGCCCTACAT CCTTCCATTG CGCCTACCCC
 17461 CGGCTATCGT GGCTACACCT ACCGCGCCAG AAGACGAGCA ACTACCCGAC CGCGAACCCAC
 17521 CACTGGAAAC CGCGCGCGCG GTGCGCGTGG CCAGCCCGTG CTGGGCCCGA TTTCGTTGCG
 17581 CAGGGTGGCT CGCGAAGGAG GCAGGACCCCT GGTGCTGCCA ACAGCGCGCT ACCACCCAG
 17641 CATGTTTAA AAGCGGTCTT TTGTGTTCT TSCAGATATG GCGCTCACTT GCGGCTCCG
 17701 TTTCGCGGTG CCGGATTTC GAGGAAGAAT GCACCGTAGG AGGGGCTAGG CCGCGCACGG
 17761 CCGTACGCGC GCGATGCGTC GTGCGACCA CCGCGCGCGC CCGCGCTGCG ACCGTGCGAT
 17821 GCGCGCGCGT ATCGTGCGCC TCCTTATTC ACTGATCGCC GCGGCGATTG GCGCGGTGCC
 17881 CGGAATTGCA TCCTGCGCCT TGCAGGCGCA GAGACACTGA TTAACAAACAA GTTGCACTGT
 17941 GAAAAATCAA AATAAAAGT CTGGACTCTC ACGCTCGCTT GGTCTCTGAA CTATTTTGT
 18001 GAATGGAGA CATCAACTTT GCGTCTCTGG CCGCGGACA CGGCTCGCGC CGGTTCTAGG
 18061 GAACTCGGCA AGATATCGGC ACCAGCAATA TGAACGCTGG CGCCTTCAGC TGGGGCTCGC
 18121 TGTGAGCGGG CATTAAAAAT TTGCGTTCCA CCGTTAAGAA CTATGGCAGC AAGGCGCTGA
 18181 ACAGCAGCAC AGGCCAGATG CTGAGGGATA AGTTGAAGAA GCAAAATTC CAACAAAGG
 18241 TGGTAGATGG CCTGGCCTCT GGCATTAGCG GGGTGGTGA CCTGGCCAAC CAGCAGTGC
 18301 AAAATAAGAT TAACAGTAAG CTTGATCCCC GCGCTCCCGT AGAGGAGCCT CCACCGCGCG
 18361 TGGAGACAGT GTCTCCAGAG GGGCGTGGCG AAAAGCGTCC GCGCCCCGAC AGGGAAGAAA
 18421 CTCTGGTGAC GCAAAATAGC GAGCCTCCCT CGTACGAGGA GGCACTAAGC CAAGGCGCTGC
 18481 CCACACCCCG TCCTCATCGC CCGATGGCTA CCGGAGTGCT GGGCCAGCAC ACACCGGTAA
 18541 CGCTGGACCT GCGTCCCCCG GCGGACACCC AGCAGAAACC TGTGCTGCCA GGGCCGACCG
 18601 CCGTGTGTGT AACCGGTCTT AGCGCGCGCT CCGTGCAGCG CCGCGCGCAG GGTCCGCGAT
 18661 CGTTGCGCGC CGTAGCCAGT GGCAACTGGC AAAGCACACT GAACAGCATC GTGGGTCTGG
 18721 GGGTGCAATC CCTGAAGGCG CGACGATGCT TCTGAATAGC TAACGTGTGG TATGTGTGTC
 18781 ATGTATGCGT CCATGTGCGC GCCAGAGGAG CTGCTGAGCC GCGCGCGCGC CGCTTTCCAA
 18841 GATGGCTACC CCTTCGATGA TGCGCGAGTG GTCTTACATG CACATCTCGG GCCAGGACCG
 18901 CTCGGAGTAC CTGAGCCCGG GCGTGGTGCA GTTTGCCCGC GCCACCGAGA CGTACTTCAG
 18961 CCTGAATAAC AAGTTTAGAA ACCCCACGCT GCGCGCTACG CACGACGTGA CCACAGACCG
 19021 GTCCAGCGCT TTGACGCTGC GGTTCATCCC TGTGGACCGT GAGGATAGTG CGTACTCGTA
 19081 CAAGGCGCGG TTCACCGTAG CTGTGGGTGA TAACCGTGTG CTGGACATGG CTTCCACGTA
 19141 CTTTGACATC CGCGCGCTGC TGGACAGGCG CCGTACTTTT AAGCCCTACT CTGGCACTGC
 19201 CTACAAACCG CTGCGTCCCA AGGGTGCCCC AAATCCTTGC GAATGGGATG AAGCTGTGAC
 19261 TGCTCTTGAA ATAAACCTAG AAGAAGAGGA CGATGACAA CAGAGCGAAG TAGACGAGCA
 19321 AGCTGAGCAG CAAAAAATC ACGTATTGAG GCAGGCGCCT TATTCTGGTA TAAATATTAC
 19381 AAAGGAGGGT ATTCAAAATG GTGTGGAAGG TCACACACCT AAATATGCCG ATAAACATT
 19441 TCAACCTGAA CCTCAAAATG GAGAATCTCA GTGTACGAA ACTGAAATTA ATCATGCAGC
 19501 TGGGAGAGTC CTTAAAAAGA CTACCCCAAT GAAACCATGT TACGGTTTAT ATGCAAAACC
 19561 CACAAATGAA AATGGAGGGC AAGGCATTCT TGTAAAGCAA CAAATGGAAG AGCTAGAAAG
 19621 TCAAGTGAAG ATGCAATTTT TCTCAACTAC TGAGGCGACC GCAGGCAATG GTGATAACTT
 19681 GACTCTTAAA GTGATATTGT ACAGTGAAGA TGTAGATATA GAAACCCGAG ACACCTCATAT
 19741 TTCTTACATG CCCACTATTA AGGAAGGTAA CTCACGAGAA CTAATGGGCC AACATCTAT

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FIGURE 21
(SHEET 6)

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19801 GCCCAACAGG CCTAATTACA TTGCTTTTAG GGACAATTTT ATTGGTCTAA TGTATTACAA
19861 CAGCACGGGT AATATGGGTG TTCTGGGGGG CCAAGCATCG CAGTTGAATG CTGTTGTAGA
19921 TTTGCAAGAC AGAAACACAG AGCTTTCATA CCAGCTTTTG CTGTATTCCA TTGGTGATAG
19981 AACCCAGTAC TTTTCTATGT GGAATCAGGC TGTGACAGC TATGATCCAG ATGTTAGAAT
20041 TATTGAAAT CATGGAAC TGATGAAC TCCAAATTAC TGCTTTCCAC TGGGAGGTGT
20101 GATTATATCA GAGACTCTTA CCAAGGTAAA ACCTAAAAA GGTGAGGAAA ATGGATGGGA
20161 AAAAGATGCT ACAGAATTTT CAGATAAAAA TGAATAAGA GTTGAAATA ATTTTGCCAT
20221 GGAATCAAT CTAAATGCCA ACCTGTGGAG AAATTTCTGT TACTCCAACA TAGCGCTGTA
20281 TTGCCCAGAC AAGCTAAAGT ACAGTCCCTC CAACGTAAAA ATTTCTGATA ACCCAACAC
20341 CTAGACTAC ATGAACAAGC GAGTGTGTGC TCCCAGGTTA GTGACTGCT ACATTAACCT
20401 TGGAGCAGCG TGGTCCCTTG ACTATATGGA CACGCTCAAC CCATTTAACC ACCACCGCAA
20461 TGCTGCGCTG CGCTACCGCT CAATGTTGCT GGGCAATGGT CGCTATGTGC CCTTCCACAT
20521 CCAGGTGCTC CAGAAGTTCT TTGCCATTAA AAACCTCCTT CTCTGCGCGG GCTCATACAC
20581 CTACGAGTGG AACCTCAGGA AGGATGTTAA CATGTTCTG CAGAGCTCCC TAGGAAATGA
20641 CCTAAGGTT CAGCGAGCCA GCATTAAGTT TGATAGCATT TGCTTTTACG CCACCTTCTT
20701 CCCCATGGCC CACAACACCG CCTCCACGCT TGAGGCCATG CTTAGAAACG ACACCAACGA
20761 CCACTCCTTT AACGACTATC TCTCCGCGCG CAACATGCTC TACCTATAC CCGCAACGC
20821 TACCAACGTT CCCATATCCA TCCCTTCCCG CAACTGGGGG GCTTTCCGCG GCTGGGCTT
20881 CAGCGCGCTT AAGACTAAGG AAACCCCATC ACTGGGCTCG GGCATCGACC CTTATTACAC
20941 CTACTCTGGC TCTATACCTT ACCTAGATGG AACCTTTTAC CTCACACACA CCTTTAAGAA
21001 GGTGGCCTAT ACCTTTGACT CTCTGTGTCG CTGGCCTGGC AATGACCGCC TGCTTACCC
21061 CAACGAGTTT GAAATTAAGC GCTCAGTTGA CGGGAGGGT TACAACGTTG CCCAGTGTA
21121 CATGACCAAA GACTGGTTCC TGGTACAAAT GCTAGCTAAC TACAACATTG GCTACCAGGG
21181 CTCTATATTC CCAGAGAGCT ACAAAGCACG CATGTACTCC TTCTTTAGAA ACTTCCAGCC
21241 CATGAGCGGT CAGGTGGTGG ATGATACTAA ATACAGGAC TACCAACAGG TGGCATCTCT
21301 ACACCAACAC AACAACTCTG GATTGTTGGT CTACCTTGCC CCCACCATCG GCGAAGGACA
21361 GGCTTACCTT GCTAACTTCC CCTATCCGCT TATAGGCAAG ACCGCAAGTG ACAGCATTAC
21421 CCAGAAAAAG TTTCTTTGGC ATCGCACCTT TTGGCGCATC CCATTCTCCA GTAACTTTAT
21481 GTCCATGGGC GCACTCAGC ACCTGGGCCA AAACCTTCTC TACGCCAACT CCGCCACGCG
21541 GCTAGACATG ACTTTTGAGG TGGATCCCAT GGACGAGCCC ACCCTTCTTT ATGTTTGTGT
21601 TGAAGTCTTT GACGTGTGCC GTGTGCACCG GCGCCACCGG GCGGTCTATG AAACCGTGTA
21661 CCTGCGCAGC CCTTCTCTGG CCGGCAACCG CACAACATAA AGAAGCAAGC AACATCAACA
21721 ACAGCTGCGG CCATGGGCTC CAGTGAGCAG GAACTGAAG CCATTTGTCA AGATCTTGGT
21781 TGTGGGCCAT ATTTTGTGG CACCTATGAC AAGCGCTTTC CAGGCTTTGT TTCTCCACAC
21841 AAGCTCGCCT GCGCCATAGT CAATACGGCC GGTGCGGAGA CTGGGGCGGT ACATGAGATG
21901 GCCTTTGCTT GGAACCGCA CTCAAAAACA TGCTACCTCT TTGAGCCCTT TGGCTTTTCT
21961 GACCAAGGAC TCAAGCAGGT TTACAGTTT GAGTACGAGT CACTCTCGCG CGTAGCGGCC
22021 ATTGCTTCTT CCCCAGCGG CTGTATAACG CTGGAAAAGT CCACCCAAAG CGTACAGGGG
22081 CCCAAGCTGG CGGCTGTGG ACTATTCTGC TGCAATGTTT TCCAAGCCTT TGCCAAGTGG
22141 CCCCAACTC CCATGGATCA CAACCCACCC ATGAACCTTA TTACCGGGGT ACCCAACTCC
22201 ATGCTCAACA GTCCCCAGGT ACAGCCACCC CTGCGTGCCA ACCAGGAACA GCTCTACAGC
22261 TTCTTGAGGC GCCACTCGCC CTACTTCCGC AGCCACAGTG CGCAGATTAG GAGCGCCACT
22321 TCTTTTGTCT ACTTGAAAAA CATGTAAAAA TAATGTACTA GAGACACTTT CAATAAAGGC
22381 AAATGCTTTT ATTTGTACAC TCTCGGGTGA TTATTACCC CCACCCCTTG CGTCTGCGCC
22441 GTTTAAAAAT CAAAGGGGTT CTGCGCGGCA TCGCTATGCG CCACTGCGAG GGACACGTTG
22501 CGTACTGCTT GTTTAGTGCT CCACCTAAAC TCAGGCACAA CCATCCGCGG CAGCTCGGNG
22561 AAGTTTTTAC TCCACAGGCT GCGCACCATC ACCAAAGCGT TTAGCAGGTC GGGCGCGGAT
22621 ATCTTGAAGT CGCAGTTGGG GCCTCCGCCC TGCGCGCGCG AGTTGCGATA CACAGGGTTG
22681 CAGCACTGGA ACACTATCAG CGCGGGTGG TGCAAGCTGG CCAGCACGCT CTTGTGAGG
22741 ATCAGATCCG CGTCCAGGTC CTCGCGGTTG CTCAGGGCGA ACGGAGTCAA CTTTGTAGC
22801 TGCTTTCCCA AAAAGGGGCG GTGCCAGGCG TTTGAGTTGC ACTCGCACCG TAGTGGCATC
22861 AAAAGGTGAC CGTCCCGGCT CTGGCGGTTA GGATACAGCG CCTGCATAAA AGCCTTGATC
22921 TGCTTTAAAG CCACCTGAGC CTTTGGGCTT TCAGAGAAGA ACATGCCGCA AGACTTGCGG
22981 GAAAACGTAT TGGCCGAGCA GGGCGCGTGC TGCAAGCAGC ACCTTGCGTC GGTGTTGGAG
23041 ATCTGCACCA CATTTCCGCC CCACCGGTTT TTCAGGATCT TGCGCTTGCT AGACTGCTCC
23101 TTCAAGCGCG GCTGCCGCTT TTCGCTCGTC ACATCCATTT CAATCACGTC CTCTTTATTT
23161 ATCATAATGC TTCCGTGTAG ACACCTAAGC TCGCTTCGA TCTCAGCGCA GCGGTGACG

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FIGURE 21
(SHEET 7)

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23221 CACAACGCGC AGCCCGTGGG CTCGTGATGC TTGTAGGTCA CCTCTGCAAA CGACTGCAGG
23281 TACGCTTGCA GGAATCOCCT CATCATCGTC ACAAAGGTCT TGTGCTGGT GAAGGTGAGC
23341 TGCAACCCGC GGTGCTCCTC GTTCAGCCAG GTCTTGCTATA CGCCCGCCAG AGCTTCCACT
23401 TGGTCAGGCA GTAGTTTGAA GTTCGCTTTT AGATCGTTAT CCACGTGGTA CTTGTCCATC
23461 AGCGCGCGCG CAGCCTCCAT GCGCTTCTCC CACGCAGACA CGATCGGCAC ACTCAGCGGG
23521 TTATCAGCG TAATTTCACT TTCCGCTTCG CTGGGCTCTT CCTCTTCTC TTGCGTCCGC
23581 ATACCAACGG CCACTGGGTC GTCTTCATT AGCCGCGCA CTGTGCGCTT ACCTCCTTG
23641 CCATGCTTGA TTAGCAGCG TGGTTGCTG AAACCCACCA TTGTAGGCG CACATCTTCT
23701 CTTTCTTCTT CCGTGTCCAC GATTACCTCT GGTATGGCG GCGCTCGGG CTTGGGAGAA
23761 GGGCGCTTCT TTTTCTTCTT GGGCGCAATG GCCAAATCCG CGCCCGAGGT CGATGGCGCG
23821 GGGCTGGTTC TGCGCGGCAC CAGCGCGTCT TGTGATGAGT CTTCTCTCTC CTGCGACTCG
23881 ATACGCGGCC TCATCGCTT TTTTGGGGGC GCGCGGGGAG GCGCGGGGGA CGGGGACGGG
23941 GAGGACAGCT CCTCCATGTT TGGGGGACGT CCGCGCGCAC CGCTTCCGCG CTGCGGGGTG
24001 GTTTCGCGCT GCTCCTCTTC CCGACTGGCC ATTTCTTCTT CCTATAGGCA GAAAAAGATC
24061 ATGAGTTCAG TCGAGAAGAA GGACAGCCTA ACCGCCCTCT CTGAGTTGCG CACCAACCGC
24121 TCCACCGATG CCGCCAAACG GCCTACACCC TTCCCGCTCG AGGCACCCCG CTTGGAGGAG
24181 GAGGAAGTGA TTATCGAGCA GGACCCAGGT TTGTGAAGCG AAGCAACGGA GGACCGCTCA
24241 GTACCAACAG AGGATAAAAA CCAAGACCCG GACAAACGCA AAGCAACGGA GGAACAAGTC
24301 GGGCGGGGGG ACGAAAGGCA TGGCGACTAC CTAGATGTGG GAGACGAGCT CTTGTGAAG
24361 CATCTGAGCG GCGAGTGGCG CATTATCTGC GACGCGTTGC AAGAGCGCAG CGATGTGCCG
24421 CTGCGCTTAG CCGATGTGAG CTTTGCTTAC GAAAGCGCAC TATTCTCACC GCGCGTACCC
24481 CCCAAACGCC AAAAAACGG CACATGCGAG CCAACCCCGC GCCTCAACTT CTACCCCGTA
24541 TTTGCGGTGC CAGAGGTGCT TGCCACCTAT CACATCTTTT TCCAAAACCT CAAGATACCC
24601 CTATCTGCGC GTGCCAACCG CAGCGGAGCG GACAAAGCAG TGGCCTTGGC GCAGGGGCGT
24661 GTCATACCTG ATATCGCCTC GCTCAACGAA GTGCCAAAAA GCGAAAATGA AAGTCACTCT
24721 GACGAGAGAG GCGCGGCAAA CGCTCTGCAA CAGGAAAACA GCGAAAATGA AAGTCACTCT
24781 GAGGTGTGGG TGAAGCTGCA GGGTGACAA CCGCGCTTAG CCGTACTAAA ACGCAGCATC
24841 GAGGTCAACC ACTTTGCTTA CCGCGCACTT AACCTACCCC CCAAGGTGAT GAGCAGATC
24901 ATGAGTGAGC TGATCGTGG CCGTGCGCAG CCGCTGGAGA GGGATGCAAA TTGTCAAGAA
24961 CAAACAGAGG AGGCGCTACC CGCAGTTGGC GACGAGCAGC TAGCGCGCTG GCTTCAAAAG
25021 CCGGAGCCTG CCGACTTGA GAGCGGACCG AAATTAATGA TGGCGCGAGT GCTCGTTACC
25081 GTGAGCTTG AGTGATGCA TCGACAGGGC TACGTACGCC AGGCCTGCAA GATCTCCAC
25141 GAAACATTGC ACTACACCTT CTCTACCTT GGAATTTTGC ACGAAAACCG CTTGGGCAA
25201 GTGAGCTCT GCAACCTGGT CTCTACCTT GGAATTTTGC ACGAAAACCG CTTGGGCAA
25261 AACGTGCTTC ATTCCACGCT CAAGGGCGAG GCGCGCGCGC ACTACGTCCG CGACTGCGTT
25321 TACTTATTTT TATGCTACAC CTGGCAGACG GCGCTGGCGC TTGGGCAACA GTGCTTGGAG
25381 GAGTGCAACC TCAAGGAGCT GCAGAAACTG CTAAAGCAAA ACTTGAAGGA CCTATGGACG
25441 GCCTTCAACG AGCGCTCCGT GCGCGCGCAC CTGGCGGACA TCAATTTTCC CGAACGCGCT
25501 CTTAAACCCC TGCAACAGGG TCTGCGAGC TTCAACAGTC AAAGCATGTT GCAGAACTTT
25561 AGGAACCTTA TCCTAGAGCG CTCAGGAATC TTGCGCGCCA CCGCTGTGTC ACTTCTTAGC
25621 GACTTTGTGC CCATTAAGTA CCGCGAATGC CCGCGCGCGC TTTGGGGCCA CTGCTACCTT
25681 CTGAGCTAG CCAACTACCT TGCGTACCAC TCTGACATA TGAAGACCT GAGCGGTGAC
25741 GGTCTACTCG AGTGTCACTG TCGCTGCAAC CTATGACCCC CGCACCGCTC CCGGTGTTGC
25801 AATTGCGAGC TGCTTAACGA AAGTCAAAAT ATCGGTACCT TTGAGCTGCA GGGTCCCTCG
25861 CTTGACGAAA AGTCCGCGCG TCGGGGCTTG AAATCTCACT CCGGGCTGTG GAGCTCGGCT
25921 TACCTTCCGA AATTGTGACC TGAGGACTAC CAGCGCCACG AGATTAGGTT CTACGAAGAC
25981 CAATCCCGCC CGCCAAATGC GCGGCTTACC GCGTGGCTCA TTACCCAGGG CCACATCTTT
26041 GGCAATTTGC AAGCCATCAA CAAAGCCCGC CAAGAGTTTC TGCTACGAAA GGGACGCGGG
26101 GTTTACTTGG ACCCCAGTTC CCGCGAGGAG CTCAACCCAA TCCCGCCGCC GCGCGAGCCC
26161 TATCAGCAGC AGCCGCGGGC CTTGTCTTCC CAGGATGGCA CCGAAAAGA AGCTGCGAGT
26221 GCGCGCGCCA CCCACGGAGC AGGAGGAATA CTGGGACAGT CAGGAGAGG AGGTTTGGGA
26281 CGAGGAGGAG GAGGACATGA TGAAGACTG GAGAGGCTTA GACGAGGAG CTTCCGAGGT
26341 CGAAGAGGTG TCAGACGAAA CACCGTCAAC CTGCGTCCGA TTCCCTCTGC CCGCGCCCCA
26401 GAAATCGGCA ACCGTTTCCA GCATGGCTAC AACCTCCGCT CCTCAGGCGC CCGCGGCACT
26461 GCGCGTTCGC CGACCCAAAC GTAGATGGGA CACCACTGGA ACCAGGGCGG GTAAAGTCCAA
26521 GCAGCGCGCG CCGTTAGCCC AAGAGCAACA ACAGCGCCAA GGCTACCGCT CATGGCGCGG
26581 GCACAAGAAC GCCATAGTTG CTGTCTTGCA AGACTGTGGG GGCACATCT CTTCCGCGCG

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FIGURE 21
(SHEET 8)

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26641 CCGCTTTCTT CTCTACCATC ACGGCGTGGC CTTCCCCCGT AACATCCTGC ATTACTACCG
26701 TCATCTCTAC AGCCCATACT GCACCGGCGG CAGCGGCAGC GGCAGCAACA GCAGCGGCCA
26761 CACAGAAGCA AAGGCGACCG GATAGCAAGA CTCTGACAAA GCCCAAGAAA TCCACAGCGG
26821 CGGAGGAGC AGGAGGAGGA GCGCTGCGTC TGCGGCCCAA CGAACCCGTA TCGACCCGCG
26881 AGCTTAGAAA CAGGATTTTT CCGACTCTGT ATGCTATATT TCAACAGAGC AGGGGCCAAG
26941 AACAGAGCT GAAAATAAAA AACAGGTCTC TGCGATCCCT CACCCGCAGC TGCGTGTATC
27001 ACAAAGCGA AGATCAGCTT CGGCGCACGC TGGAGAGCGC GGAGGCTCTC TTCAGTAAAT
27061 ACTGGCGCGT GACTCTTAAG GACTAGTTTC GCGCCCTTTC TCAAAATTAA GCGCGAAAAC
27121 TAGTCACTCT CCAGCGGCCA CACCCGCGCG CAGCACCTGT CGTCAGCGCC ATTATGAGCA
27181 AGGAAATTC CACGCCCTAC ATGTGGAGTT ACCAGCCACA AATGGGACTT CCGCGTGGAG
27241 CTGCCCCAAGA CTACTCAACC CGAATAAACT ACATGAGCGC GGGACCCACC ATGATATCCC
27301 GGGTCAACGG AATCCGCGCC CACCGAAACC GAATTCTCTT GGAACAGCGG GCTATTACCA
27361 CCACAGCTCG TAATAACCTT AATCCCCGTA GTTGGCCCGC TGCCCTGGTG TACCAGGAAA
27421 GTCCCGCTCC CACCACTGTG GTACTTCCCA GAGACGCCCA GGCAGAGTT CAGATGACTA
27481 ACTCAGGCGC CGAGCTTGGG GCGCGCTTTC GTACAGGGT GCGGTCGCC GGGCAGGGTA
27541 TAATCACTCT GACAATCAGA GGGCGAGGTA TTCAGCTCAA CGAGAGTGG GTGAGCTCCT
27601 CGCTTGGTCT CGTCCGAGAC GGGACATTTC AGATCGCGCG CGCCGCGCCT CTTTCAATCA
27661 CGCTCGTCA GGAATCTCTA ACTCTGCAGA CCGTCTCTCT TGAGCCGCGC TCTGGAGGCA
27721 TTGGAACTCT GCAATTTATT GAGGAGTTTG TGCCATCGGT CTACTTTAAC CCCTTCTCGG
27781 GACCTCCCGG CCACTATCCG GATCAATTTA TTCTAACTT TGACGCGGTA AAGGACTCGG
27841 CGGAGCGGTA CGACTGAATG TTAAGTGGAG AGGCAGAGCA ACTGCGCCTG AAACACTCGG
27901 TCCACTGTCC CGGCCACANG TGCTTTGCCG GCGACTCCGG TGAGTTTTCG TACTTTGAAT
27961 TGCCCGAGGA TCATATCGAG GCGCGGCGC CCGGCTCCG GCTTACGCC CAGGAGAGGC
28021 TTGCCGCTAG CCGATTCCGG GAGTTTACCC AGCGCCCTCT GCTAGTTGAG CGGACAGGG
28081 GACCCCTGTG TCTCACTGTG ATTTGCAACT GTCTTAACCT TGGATTACAT CAAGATCTTT
28141 GTTCCCATCT CTGTGCTGAG TATAATAAAT ACAGAAATTA AATATACTG GGGCTCTAT
28201 CGCCATCCCT TAAACGCCAC CGTCTTCAAC CGCCCAAGCA AACCAAGCGG AACCTTACCT
28261 GGTACTTTTA ACATCTCTCC CTCTGTGATT TACAACAGTT TCAACCCAGA CGGAGTGAGT
28321 CTACGAGAGA ACCTCTCCGA GCTCAGCTAC TCCATCAGAA AAAACACCAC CCTCTTACC
28381 TGCCCGGAAC GTACGAGTGC GTACCGGCC GCTGCACCAC ACCTACGCC TGACCGTAAA
28441 CCAGACTTTT TCCGGACAGA CCTCAATAAC TCTGTTTACC AGAACAGGAG GTGAGCTTAG
28501 AAAACCCCTA GGGTATTAGG CCAAAGGCGC AGCTACTGTG GGGTTTATGA ACAATTCAG
28561 CAACTCTACG GGCTATTCTA ATTCAGGTTT CTCTAGAATC GGGGTTGGGG TTATTCTCTG
28621 TCTTGATGAT CTCTTTATTC TTATACTAAC GCTTCTCTCG CTAAGGCTCG CCGCTGCTG
28681 TGTGCACATA TGCTTTTATT GTCACTTTT TAAACGCTGG GGTCCGCCAC CAAGATGATT
28741 AGGTACATAA TCCTAGGTTT ACTCACCTT GCGTCAGCCC ACGGTACCAC CCAAAGGTTG
28801 GATTTTAAAG AGCCAGCCTG TAATGTTACA TTGCGAGCTG AAGCTAATGA GTGCACCACT
28861 CTTATAAAAT GCACCACAGA ACATGAAAG CTGCTTATTC GCCACAAAA CAAAATGCG
28921 AAGTATGCTG TTTATGCTAT TTGGCAGCCA GGTGACACTA CAGAGTATAA TGTACAGTT
28981 TTCCAGGGTA AAAGTCATAA AACTTTTATG TATACTTTTC CAITTTATGA AATGTGCGAC
29041 ATTACCATGT ACATGAGCAA ACAGTATAAG TTGTGGCCCC CACAAAATG TGTGAAAAC
29101 ACTGGCACTT TCTGCTGCAC TGCTATGCTA ATTACAGTGC TCGCTTTGGT CTGTACCCTA
29161 CTCTATATTA AATACAAAAG CAGACGCGAG TTTATTGAGG AAAAGAAAAA GCTTTAATTT
29221 ACTAAGTTAC AAAGCTAATG TCACCACTAA CTGCTTTACT CGCTGCTTGC AAAACAAATT
29281 CAAAAGTTA GCMTTATAAT TAGAATAGGA TTTAAACCCC CCGGTCAATT CCTGCTCAAT
29341 ACCATTCCCC TGAACAATG ACTCTATGTG GGATATGCTC CAGCGCTACA ACCTTGAAAT
29401 CAGGCTTCCCT GGATGTCAGC ATCTGACTTT GCGCAGCACG TGTCCCGCGG ATTTGTTCOA
29461 GTCCAACTAC AGCGACCCAC CCTACAGAG ATGACCAACA CAACCAAGCG GCGCGCGGCT
29521 ACCGCACTTA CATCTACCAC AAATACACCC CAAGTTTCTG CCTTTGTCAA TAACCTGGAT
29581 AACTTGGGCA TGTGTTGGTT CTCCATAGCG CTTATGTTTG TATGCCCTAT TATTATGTTG
29641 CTCATCTGCT GCCTAAAGCG CAAACGCGCC CGACCAACCA TCTATAGTCC CATCAATTG
29701 CTACACCCAA ACAATGATGG AATCCATAGA TTGGACGGAC TGAACACAT GTTCTTTTCT
29761 CTTACAGTAT GATTAAATGA GACATGATTC CTCGAGTTT TATATTACTG ACCCTTTGTTG
29821 CGCTTTTTTG TGCGTGTCTC ACATTGCGTGG CCGTTTCTCA CATCGAAGTA GACTGCTATC
29881 CAGCCTTAC AGTCTATTG CTTTACGGAT TTGTACCCCT CACGCTCATC TGCAGCTCA
29941 TCACTGTGCT CATCGCCTT ATCCAGTGCA TTGACTGGGT CTGTGTGCGC TTTGCATATC
30001 TCAGACACCA TCCCAGTAC AGGACAGGA CTATAGCTGA GCTTCTTAGA ATTCTTTAAT

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FIGURE 21
(SHEET 9)

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30061 TATGAATTT ACTGTGACTT TTCTGCTGAT TATTTGCACC CTATCTGCGT TTTGTTCCCC
30121 GACCTCCAAG CCTCAAAGAC ATATATCATG CAGATTCACG CGTATATGGA ATATTCCAAG
30181 TTGCTACAAAT GAAAAAGCG ATCTTTCCGA AGCCTGGTTA TATGCAATCA TCTCTGTTAT
30241 GGTGTTCTGTC AGTACCATCT TAGCCCTAGC TATATATCCC TACCTTGACA TTGGCTGGAA
30301 ACGAATAGAT GCCATGAACC ACCCAAATTT CCCCGCGCCC GCTATGCTTC CACTGCAACA
30361 AGTTGTTGCC GCGGCTTTG TCCAGGCCAA TCAGCCTCGC CCCACTTCTC CCACCCCCAC
30421 TGAATTCAGC TACTTTAATC TAACAGGAGG AGATGACTGA CACCCTAGAT CTAGAAATGG
30481 ACGGAATAT TACAGAGCAG OGCTGTAG AAAGACGAG GGCAGCGGCC GAGCAACAGC
30541 GCATGAATCA AGAGCTCCAA GACATGGTTA ACTTGACCA GTGCAAAAGG GGTATCTTTT
30601 GTCTGGTAAA GCAGGCCAAA GTCACCTACG ACAGTAATAC CACGGACAC CGCCTTAGCT
30661 ACAAGTTGCC AACCAAGCGT CAGAAATTGG TGGTCATGGT GGGAGAAAG CCCATTACCA
30721 TAATCTCAGCA CTCGGTAGAA ACCGAAGGCT GCATTCACCT ACCTTGTCAA GGACCTGAGG
30781 ATCTCTGACG CTTTATTAAG ACCCTGTGCG GTCTCAAAGA TCTTATTCCC TTTAACTAAT
30841 AAAAAAAT AATAAGCAT CACTTACTTA AAATCAGTTA GCAAAATTCT GTCCAGTTTA
30901 TTCAGCAGCA CCTCCTGCC CTCTCCAG CTCTGGTATT GCAGCTTCTT CTTGGCTGCA
30961 AACTTTCTCC ACAATCTAAA TGGAAATGCA GTTCTCTCT GTTCTGTCC ATCCGCAACC
31021 ACTATCTCA TGTGTGTGCA GATGAAGCGC GCAAGACCGT CTGAAGATAC CTTCAACCCC
31081 GTGTATCCAT ATGACAGCGA AACCGTCTCT CCAACTGTGC CTTTCTTAC TCCTCCTTTT
31141 GTATCCCCCA ATGGGTTTCA AGAGAGTCCC CCTGGGGTAC TCTCTTTGCG CCTATCCGAA
31201 CCTCTAGTTA CTTCAATGG CATGCTTGGC CTCAAAATGG GCAACGCGCT CTCTCTGGAG
31261 GAGGCCGCGA ACCTTACCTC CCAAAATGTA ACCACTGTGA GCCCACTCTT CAAAAAACC
31321 AAGTCAACCA TAAACCTGGA AATATCTGCA CCCCCTACAG TTACCTCAGA AGCCCTAAT
31381 GTGGCTGCGC CGCACCTCT AATGGTCGCG GGCACACAC TCACCATGCA ATCAGAGGCC
31441 CGGCTAACCG TGACGACTC CAACTTAGC ATTGCCACCC AAGGACCCCT CACAGTGTCA
31501 GAGGAAAGC TAGCCCTGCA AACATCAGGC CCCCACCA CCACGATAG CAGTACCCCT
31561 ACTATCACTG CCTCACCCC TCTAACTACT GCCACTGGTA GCTTGGGCAT TGACTGAAA
31621 GAGCCCATTT ATACACAAA TGGAAACTA GCACTAAAGT ACGGGCTCTT TTTGATGTA
31681 ACAGACGACC TAAACACTTT GACCGTAGCA ACTGGTCCAG GTGTGACTAT TAATAATCT
31741 TCCTTGACAA CTAAAGTTAC TGGAGCCTTG GGTTTTGATT CACAAGGCAA TATGCAACTT
31801 AATGTAGCAG GAGGACTAAG GATTGATTCT CAAAACAGAC GCCTTATACT TGATGTTAGT
31861 TATCCGTTTG ATGCTCAAAA CCACTAAAT CTAAGACTAG GACAGGGCCC TCTTTTATA
31921 AACTCAGCCC ACAACTTGA TATTAACTAC AACAAAGGCC TTTACTTGT TACAGCTTCA
31981 AACAAATCCA AAAAGCTTGA GGTAAACCTA AGCACTGCCA AGGGGTGTAT GTTTGACGCT
32041 ACAGCCATAG CCATTAATGC AGGAGATGGG CTTGAATTTG GTTCACCTAA TGCACCAAAC
32101 ACRAATCCCC TCAAAACAAA AATTGGCCAT GGCCTAGAAT TTGATTCAAA CAAGGCTATG
32161 GTTCTTAAC TAGGACTGG CCTTAGTTT GACAGCACAG GTGCCATTAC AGTAGGAAAC
32221 AAAAAATAAG ATAAGCTAAC TTTGTGGACC ACACCAGTC CATCTCCTAA CTGTAGACTA
32281 AATGCAGAGA AAGATGCTAA ACTCACTTTG GTCTTAACAA AATGTGGCAG TCARAATCTT
32341 GCTACAGTTT CAGTTTGGC TGTAAAGGC AGTTTGGCTC CAATATCTGG AACAGTTCAA
32401 AGTGCTCATC TTATTATAAG ATTTGACGAA AATGGAGTGC TACTAAACAA TTCCCTCTCG
32461 GACCCAGAT ATTGGAACCT TAGAATGGA GATCTTACTG AAGGCACAGC CTATACAAAC
32521 GCTGTGGAT TTATGCCATA CCTATCAGCT TATCCAAAT CTCAAGTAA AACTGCCAAA
32581 AGTAACATTG TCAGTCAAGT TTAATTAAAC GGAGACAAA CTAACCTGT AACTAACC
32641 ATTACACTAA ACGGTACACA GGAACAGGA GACACAATC CAAGTGATA CTCTATGTCA
32701 TTTTCAATGG ACTGGTCTGG CCACAATAC ATTAATGAAA TATTGGCCAC ATCCTCTTAC
32761 ACTTTTTCAT ACATTGCCCA AGAATAAAGA ATCGTTTGT TTTATGTTCA ACGTGTAT
32821 TTTTCAATGG CAGAAAATTT CAAGTCATTT TTCAATCAGT AGTATAGCCC CACCAACCA
32881 TAGCTTATAC AGATCACCGT ACCTTAATCA AACTCACAGA ACCCTAGTAT TCAACCTGCC
32941 ACCTCCCTCC CAACACAG AGTACACAGT CCTTCTCCC CGGCTGGCT TAAAAAGCAT
33001 CATATCATGG GTAACAGACA TATTCTTAGG TGTATATTG CACACGGTTT CCTGTGAGC
33061 CAAACGCTCA TCAGTGATAT TAATAAACTC CCGGGGAGC TCACCTAAGT TCATGTGCT
33121 GTCCAGCTGC TGAGCCACAG GCTGCTGTCC AACTTGGGT TGCTTAAAG GCGCGAAGG
33181 AGAAGTCCAC GCCTACATGG GGGTAGAGTC ATAATCGTGC ATCAGGATAG GCGGTGGT
33241 CTGACAGC GCGCAATAA ACTGCTGCGC CCGCGCTCC GTCCGTGAGG AATACACAT
33301 GGCAGTGGTC TCCTCAGCGA TGATTGCGAC CGCCGCGAGC ATAAGGCGCC TTGCTCTCG
33361 GGCACAGC GGCACCTGA TCTCACTTAA ATCAGCACAG TAACTGCAGC ACAGCACCAC
33421 AATATTGTTT AAAATCCCAG AGTGCAAGGC GCTGTATCCA AAGCTCATGG CCGGAGCCAC

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FIGURE 21
(SHEET 10)

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33481 AGAACCACG TGGCCATCAT ACCACAAGCG CAGGTAGATT AAGTGGGAC CCCTCATAAA
33541 CACGCTGGAC ATAAACATTA CCTCTTTTGG CATGTTGTAA TTCACCACCT CCCGGTACCA
33601 TATAAACCTC TGATTAAACA TGGGCGCATC CACCACCATC CTAACCACG TGGCCAAAAC
33661 CTGCCCGCG GCTATACACT GCAGGGAACC GGGACTGGAA CAATGACAGT GGAGAGCCCA
33721 GGACTCGTAA CCATGGATCA TCATGCTCOT CATGATATCA ATGTGGCAC AACACAGGCA
33781 CACGTGCATA CACTTCCTCA GGATTACAAG CTCCTCCCGC GTTAGAACCA TATCCAGGG
33841 AACCAACCAT TCCTGAATCA GCGTAAATCC CACACTGCAG GGAAGACCTC GCACGTAACT
33901 CACGTTGTGC ATTGTCAAAG TGTATCATTC GGGCAGCAGC GGATGATCCT CCAGTATGGT
33961 AGCGCGGGTT TCTGTCTCAA AAGGAGGTAG ACGATCCCTA CTGTACGGAG TGGCCGAGA
34021 CAACCGAGAT CGTGTGGTC GTAGTGTCT GCGAAATGGA ACGCCGACG TAGTCATATT
34081 TCCTGAAGCA AAACCAAGTG CCGGCGTGAC AAACAGATCT GCGTCTCCGG TCTCGCCGCT
34141 TAGATCGCTC TGTGTAGTAG TTGTAGTATA TCCACTCTCT CAAAGCATCC AGGCGCCGCC
34201 TGGCTTCGGG TTCTATGTAA ACTCCTTCAT CGCGCGCTGC CCTGATAACA TCCACCACCG
34261 CAGAAATAGC CACACCAGC CAACCTACAC ATTCTTCTG CGAGTCACAC ACGGAGGAG
34321 CGGGAAGAGC TGGAAAGAAC ATGTTTTTTT TTTTATTCCA AAAGATTATC CAAACCTCA
34381 AAATGAAGAT CTATTAAGTG AACCGGCTCC CCTCCGGTGG CGTGGTCAA CTCTACAGCC
34441 AAAGACAGA TAATGGCATT TGTAAAGTGT TGCACATGG CTTCACAAAG GCAACCGCC
34501 CTCACGTCCA AGTGAAGTA AAGGCTAAAC CCTCAGGGT GAATCTCTC TATAAACATT
34561 CCAGCACCTT CAACCATGCC CAAATAATTC TCATCTCGCC ACCTTCTCAA TATATCTCTA
34621 AGCAATCCG GAATATTAA TCCGGCCATT GTAAAAATCT GCTCCAGAGC GCGCTCCACC
34681 TTCAGCTCA AGCAGCGAAT CATGATTGCA AAAATTCAAG TTCTCACAG ACCTGTATAA
34741 GATTCAAAGC CGGAACATTA ACAAAATAAC CGCGATCCCG TAGGTCCCTT CGCAGGCCA
34801 GCTGAACATA ATCGTGCAGG TCTGCACGGA CCAGCGCGGC CACTTCCCGC CCAGGAACCT
34861 TGACAAAAGA ACCCACTG ATTAGACAC GCATACCTGG AGCTATGCTA ACCAGCGTAG
34921 CCCGATGTA AGCTTTGTGT CATGGCGGC GATATAAAAT GCAAGGTGCT GCTCAAAAAA
34981 TCAGGCAAG CCTCGCGCAA AAAAGAAAGC ACATCGTAGT CATGCTCATG CAGATAAAGG
35041 CAGGTAAGCT CCGGAACAC CACAGAAAAA GACACCATTT TTCTCTCAA CATGCTCGG
35101 GGTTCCTGCA TAAACACAAA ATAAATAAC AAAAAACAT TTAACATTA GAAGCCTGTC
35161 TTACAAACAG AAAACAACC CTTATAAGCA TAAGACGGAC TACGGCCATG CCGCGGTGAC
35221 CGTAAAAAAA CTGGTCACCG TGATTAAAAA GCACCAACGA CAGCTCCTCG GTCATGTCCG
35281 GAGTCATAAT GTAAGACTCG GTAAACACAT CAGGTTGATT CATCGGTGAG TGCTAAAAAG
35341 CGACCGAAAT AGCCCGGGGG AATACATACC CGCAGGCGTA GAGACAACAT TACAGCCCCC
35401 ATAGGAGTA TAACAAAATT AATAGGAGAG AAAACACAT AAACACCTGA AAACCCCTCC
35461 TGCTAGGCA AATAGCACC CTCCTCTCC AGAACACAT ACAGCGCTTC ACAGCGGACG
35521 CCTAACAGTC AGCCTTACCA GTAAAAAGA AAACCTATTA AAAAAACCC ACTCGACAG
35581 GCACACGTC AATCAGTCAC AGTGTAAAAA AGGCGCAAGT GCAGAGGAG TATATATAGG
35641 ACTAAAAAAT GACGTAAOAG TTAAGTCCA CAAAAACAC CCAGAAAAAC GCACGGAAC
35701 CTACGCCAG AAACGAAAGC CAAAAACCC ACAACTTCT CAAATCTGCA CTTCGGTTTT
35761 CCCAGTTAC GTAACCTTCC ATTTTAAAG AAACACAT CCACACAT ACAAGTTACT
35821 CCGCCCTAAA ACCTAGTCA CCGCCCGGT TCCACGCCG CGCGCCAGT CACAACTCC
35881 ACCCCCTCAT TATCATATTG GCTTCATCC AAAATAAGGT ATATTATTGA TGATG

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FIGURE 21
(SHEET 11)

ad5

11

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LOCUS KD1 33592 bp DNA SYN 28-APR-1999
DEFINITION KD1
ACCESSION KD1
KEYWORDS .
SOURCE Unknown.
ORGANISM Unknown
Unclassified.
REFERENCE 1 (bases 1 to 33592)
AUTHORS Self
JOURNAL Unpublished.
FEATURES
CDS Location/Qualifiers
1..33592
/gene="KD1"
/product="KD1"
BASE COUNT 7744 a 9470 c 9285 g 7093 t
ORIGIN
1 CATCATCAAT AATATACCTT ATTTTGGATT GAAGCCAATA TGATAATGAG GGGGTGGAGT
61 TTGTGACGTG GCGCGGGGCG TGGGAACGGG GCGGGTGACG TAGTAGTGTG GCGGAAGTGT
121 GATGTTGCAA GTGTGGCGGA ACACATGTAA GCGACGGATG TGGCAAAAGT GACGTTTTTG
181 GTGTGCGCGG GTGTACACAG GAAGTGACAA TTTTCGCGCG GTTTTAGGCG GATGTTGTAG
241 TAAATTTTGG CGTAACCGAG TAAGATTTCG CCATTTTCGC GGGAAAACGT AATAAGAGGA
301 AGTGAATCT GAATAATTTT GTGTTACTCA TAGCGCGTAA TATTGTCTA GGGCGCGGG
361 GACTTTGACC GTTTACGTGG AGACTCGCCC AGGTGTTTTT CTCAGGTGTT TTCGCGCTTC
421 CCGGTCAAAG TTGGCGTTTT ATTATTATAG TCAGCTGACG TGAGTGTAT TTATACCCGG
481 TGAGTTCCTC AAGAGGCCAC TCTTGAGTGC CAGCGAGTAG AGTTTTCTCC TCCGAGCCGG
541 TCCGACACCG GGACTGAAA TGAGACATGA GGTACTGGCT GATAATCTTC CACCTCCTAG
601 CCATTTTGAA CCACCTACCC TTCAAGAACT GTATGATTTA GACGTGACGG CCCCGAAGA
661 TCCCAACGAG GAGCGCGTTT CCGAGATTTT TCCCGACTCT GTAATGTTGG CCGTGCAGGA
721 AGGGATTGAC TTACTCACTT TTCGCGCGCG CCGCGGTCTC CCGGAGCCGC CTCACCTTTC
781 CCGGCAGCCC GAGCAGCGCG AGCAGAGAGC CTGGGTCCG GTTTGCCACG AGGCTGGCTT
841 TCCACCCAGT GACGACGAGG ATGAAGAGGG TGAGGAGTTT GTGTTAGATT ATGTGGAGCA
901 CCGCGGCCAC GGTTCAGGT CTGTCAATTA TCACCGGAGG AATACGGGGG ACCCAGATAT
961 TATGTGTTCC GTTGTCTATA TGAGGACCTG TGGCATGTTT GTCTACAGTA AGTGAAATTT
1021 ATGGGCACTG GGTGATAGAG TGGTGGGTTT GGTGTGTTAA TTTTTTTTTT AATTTTTACA
1081 GTTTTGTGTT TTAAGAATT TTGTATTGTG ATTTTTTTAA AAGGTCTCTG GTCTGAACCT
1141 GAGCCTGAGC CCGAGCCAGA ACCCGAGCCT GCAAGACCTA CCGCGCTCC TAAATGGCG
1201 CCGTCTATCC TGAGACGCCC GACATCACCT GTGTCTAGAG AATGCAATAG TAGTACGGAT
1261 AGCTGTGACT CCGGTCTCTC TAACACACCT CCGTGAATAC ACCCGGTGCT CCCGCTGTGC
1321 CCCATTAAAC CAGTTGCCGT GAGAGTTGTT GGGCGTCGCC AGGCTGTGGA ATGTATCGAG
1381 GACTTGTCTA ACGAGCCTGG GCAACCTTTG GACTTGAGCT GTAAACGCCC CAGGCCATAA
1441 GGTGTAAACC TGTGATTGCG TGTGTGTTA ACGCCTTTGT TTGCTGAATG AGTTGATGTA
1501 AGTTTAAATA AGGGTGAGAT AATGTTTAA TGGCATGGCG TGTAAATGG GCGGGGCTT
1561 AAAGGGTATA TAATGCGCGG TGGGCTAATC TTGGTTACAT CTGACCTCAT GGAGGCTTGG
1621 GAGTGTTTGG AAGATTTTTT TGCTGTGCGT AACTTGCTGG AACAGAGCTC TAACAGTACC
1681 TCTTGGTTTT GAGGTTTCT GTGGGGTCA TCCGAGCAA AGTTAGTCTG CAGAATTAA
1741 GAGGATTACA AGTGGGAATT TGAAAGCTT TTGAATCCT GTGGTGAGCT GTTTGATTCT
1801 TTGAATCTGG GTACACAGGC GCTTTTCCAA GAGAAGTCA TCAAGACTTT GGATTTTTCC
1861 ACACCGGGGC GCGCTGCGGC TGCCTGTGCT TTTTGTAGTT TTATAAAGGA TAAATGGAGC
1921 GAAGAAACCC ATCTGAGCGG GGGGTACCTG CTGGATTTTC TGGCCATGCA TCTGTGGAGA
1981 GCGGTTGTGA GACACAAGAA TCGCCTGCTA CTGTTGCTT CCGTCCGCCC GGCGATAATA
2041 CCGACGGAGG AGCAGCAGCA GCGACAGGAG GAAGCCAGGC GCGCGCGGCA GGAGCAGAGC
2101 CCATGGAACC CGAGAGCCCG CCTGGACCTT CCGAATGAA TGTGTACAG GTGGCTGAAC
2161 TGTATCCAGA ACTGAGACGC ATTTTGACAA TTACAGAGGA TGGCCAGGGG CTAAAGGGGG
2221 TAAAGAGGGA GCGGGGGGCT TGTGAGGCTA CAGAGGAGGC TAGGAATCTA GCTTTTAGCT
2281 TAATGACCAG ACACCGTCTT GAGTGATTA CTTTCAACA GATCAAGGAT AATTGCGCTA
2341 ATGAGCTTGA TCTGCTGGCG CAGAAGTATT CCATAGAGCA GCTGACCACT TACTGGCTGC
2401 AGCCAGGGGA TGATTTTGAG GAGGCTATTA GGTATATGC AAAGGTGGCA CTTAGGCCAG

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FIGURE 22
(SHEET 1)

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2461 ATTGCAAGTA CAAGATCAGC AAACITGTAA ATATCAGGAA TTGTTGCTAC ATTTCTGGGA
2521 ACGGGGCCGA GGTGGAGATA GATACGGAGG ATAGGGTGGC CTTTAGATGT AGCATGATAA
2581 ATATGTGGCC GGGGGTGCTT GGCATGGACG GGGTGGTTAT TATGAATGTA AGGTTTACTG
2641 GCCCAATTT TAGGGGTACG GTTTTCTGG CCAATACCAA CCTTATCCTA CACGGTGTAA
2701 GCTTCTATGG GTTTAACAAT ACCTGTGTGG AAGCCTGGAC CGATGTAAGG GTTCGGGGCT
2761 GTGCCTTTTA CTGCTGCTGG AAGGGGGTGG TGTGTCCGCC CAAAAGCAGG GCTTCAATTA
2821 AGAAATGCCT CTTTGAAGG TGTACCTTGG GTATCCTGTC TGAGGGTAAC TCCAGGGTGC
2881 GCCACAATGT GGCCTCCGAC TGTGTTTGTCT TCATGCTAGT GAAAAGCGTG GCTGTGATTA
2941 AGCATAACAT GGTATGTGGC AACTGCGAGG ACAGGGCCTC TCAGATGCTG ACCTGCTCGG
3001 ACGGCAACTG TCACCTGCTG AAGACCATTC ACGTAGCCAG CCACCTCTCGC AAGGCTTGGC
3061 CAGTGTTTGA GCATAACATA CTGACCCGCT GTTCTTGCA TTTGGGTAAC AGGAGGGGGG
3121 TGTCTTACC TTACCAATGC AATTGTAGTC ACACTAAGAT ATTGCTTGAG CCGGAGAGCA
3181 TGTCCAGAGT GAACCTGAAC GGGGTGTTTG ACATGACCAT GAAGATCTGG AAGGTGCTGA
3241 GGTACGATGA GACCCGACAC AGGTGCAGAC CTTGCGAGTG TGGCGGTAAA CATATTAGGA
3301 ACCAGCCTGT GATGCTGGAT GTGACCGAGG AGCTGAGGCC CGATCACTTG GTGCTGGCCT
3361 GCACCCGCGC TGAAGTTTGGC TCTAGCGATG AAGATACAGA TTGAGTACTT GAAATGTGTG
3421 GCGCTGGCTT AAGGGTGGGA AAGAATATAT AAGGTGGGGG TCTTATGTAG TTTGTATCT
3481 GTTTTGCAGC AGCCGCGGCC GCCATGAGCA CCAACTCGTT TGATGGAAGC ATTGTGAGCT
3541 CATATTGAC AACCGCGCATG CCCCCATGGG CCGGGGTGCG TCGAATGTG ATGGGCTCCA
3601 GCATGTGATG TCGCCCGCTG CTGCCCGCAA ACTCTACTAC CTTGACCTAC GAGACCGTGT
3661 CTGGACCGCG GTTGGAGACT GCAGCCTCCG CCGCGCTTC AGCCGCTGCA GCCACCGCCC
3721 GCGGGATTGT GACTGACTTT GCTTTCCTGA CCGCGCTTC AAGCAGTGCA GCTTCCCGTT
3781 CATCGCGCCG CGATGACAAG TTGACGGCTC TTTTGGCACA ATTGGAITTC TTGACCGGGG
3841 AACTTAATGT CGTTTCTCAG CAGCTGTGGG ATCTGCGCCA GCAGGTTTCT GCCCTGAAGG
3901 CTTCTCTCCC TCCCAATGCG GTTTAAACA TAAATAAAA ACCAGACTCT GTTTGATTT
3961 GGATCAAGCA AGTGTCTTGC TGTCTTTATT TAGGGTTTTT GCGCGCGCGG TAGGCCCGGG
4021 ACCAGCGCTG TCGGTCTGTG AGGGTCTCTGT GTATTTTTTC CAGGACGTGG TAAAGGTGAC
4081 TCTGATGTTT CAGATACATG GGCATAAGCC CGTCTCTGGG GTGGAGGTAG CACCACCTGA
4141 GAGCTTCAATG CTGCGGGGTG GTGTTGTAGA TGATCCAGTC GTAGCAGGAG CGCTGGGCGT
4201 GGTGCTAAA AATGCTTTTC AGTAGCAAGC TGATTGCCAG GGGCAGGGCC TTGGTGTAAAG
4261 TGTTTACAAA GCGGTAAAG TGGGATGGGT GCATACGTGG GGATATGAGA TGCATCTTGG
4321 ACTGTATTTT TAGGTGCGCT ATGTTCCAG CCATATCCCT CCGGGGATTC ATGTTGTGCA
4381 GAACCCAGC CACAGTGTAT CCGGTGCACT TGCGAAATTT GTCATGTAGC TTAGAAGGAA
4441 ATGCGTGGAA GAACCTGGAG ACGCCCTTGT GACCTCCAAG ATTTTCCATG CATTCGTCCA
4501 TAATATGAGC AATGGGCCCA CCGGCGGCGG CCTGGGCGAA GATATTTCTG GGATCACTAA
4561 CGTCATATGT GTGTTCCAGG ATGAGATCGT CATAGGCCAT TTTTACAAAG CCGGGGCGGA
4621 GGTGCGCAGA CTGCGGTATA ATGTTCCAT CCGGCCCAGG GCGGTAGTTA CCCTCACAGA
4681 TTTGCATTTT CCACGCTTTG AGTTCAGATG GGGGATCAT GTCTACCTGC GGGGCGATGA
4741 AGAAAAACGGT TTCCGGGTA GGGGAGATCA GCTGGGAAGA AAGCAGGTTC CTGAGCAGCT
4801 GCGACTTACC GCAGCCCGTG GGGCCGTAAA TCACACCTAT TACCGGGTGC AACTGGTAGT
4861 TAAGAGAGCT GCAGCTGCCG TCATCCCTGA GCAGGGGGGC CACTTCGTGA AGCATGTCCC
4921 TGAATCGCAT GTTTTCCCTG ACCAAATCCG CCAGAAGGCG CTCGCGGCCC AGCGATAGCA
4981 GTTCTTGCAA GGAAGCAAAG TTTTCAACG GTTTGAGACC GTCCGCGGTA GGCATGCTTT
5041 TGAGCGTTTG ACCAAGCAGT TCCAGGCGGT CCCACAGCTC GGTCACTGCT TCTACGSCAT
5101 CTGATCCAGC CATATCTCCT CGTTTCGCGG GTTGGGGCGG CTTTCTGCTG ACGGCGTAGS
5161 TCGGTGCTCG TCCAGACGGG CCAGGGTCAT GTCTTTCCAC GGGCGCAGGG TCCTCGTCAG
5221 CGTAGTCTGG GTACAGGTGA AGGGGTGCGC TCCGGCTGCT CCGCTGCGCA GGGTGCCTT
5281 GAGGCTGTGT CTGCTGGTGG TGAAGCGCTG CCGGTCTTGG CCGTGCCTGT CCGCCAGGTA
5341 GCATTGTACC ATGTTGTGAT AGTCCAGCCC CTCGCGGCGG TGGCCCTTGG CGCGCAGCTT
5401 GCGCTTGGAG GAGGCGCGCG ACGAGGGGCA GTGCAGACTT TTGAGGCGGT AGAGCTTGGG
5461 CGCGAGAAAT ACCGATTCCG GGGAGTAGGC ATCCGCGCGC CAGGCCCCGC AGACGGTCTC
5521 GCATTCCACG AGCCAGGTGA GCTCTGGCCG TCCGGGCTCA AAAACCAAGT TTCCCCCATG
5581 CTTTGTGATG CGTTTCTTAC CTCTGTTTTT CATGAGCCGG TGTCACGCTC CGGTGAAGAA
5641 AAGGCTGTCC GTGTCCCGGT ATACAGACTT GAGAGGCTTG TCCTCGAGCG GTGTTCCCGG
5701 GTCTCTCTCG TATAGAAACT CGGACCACCT TGAGACAAAG GCTCGCGTCC AGGCCAGCAC
5761 GAAGGAGGCT AAGTGGGAGG GTTAGCGGTC GTTGTCCACT AGGGGGTCCA CTCGCTCCAG
5821 GGTGTGAAGA CACATGTGCG CCTCTTCGGC ATCAAGGAAG GTGATTGGTT TGTAGGTGTA

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FIGURE 22
(SHEET 2)

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5881 GGCCACGTGA CCGGGTGTTC CTGAAGGGGG GCTATAAAG GGGGTGGGG CGCGTTCGTC
5941 CTCACTCTCT TCCGCATCGC TGCTGCGAG GGCAGCTGT TGGGTGAGT ACTCCCTCTG
6001 AAAAGCGGGC ATGACTTCTG CGCTAAGATT GTGAGTTTCC AAAACGAGG AGGATTTGAT
6061 ATTCACTGGC CCGCGGTGA TGCCCTTGAG GGTGGCGCA TCCATCTGGT CAGAAAAGAC
6121 AATCTTTTGG TTGTCAAGCT TGGTGGCAAA CGACCCGTAG AGGGCGTTGG ACAGCAACTT
6181 GGCGATGGAG CGCAGGGTTT GGTTTTGTTC GCGATCGGCG CGCTCCTGG CGCGATGTT
6241 TAGCTGCAGC TATTGCGCGC CAACGCAACG CCATTGCGGA AAGACGGTGG TGGCTCGTC
6301 GGGCACCAGG TGCACGCGCC AACCGCGGTT GTGACGGGTG ACAAGGTCAA CGCTGGTGGC
6361 TACCTCTCCG CGTAGGCGCT CGTTGGTCCA GCAGAGGCGG CGGCCCTTGC GCGAGCAGAA
6421 TGGCGGTAGG GGGTCTAGCT GCGTCTCGTC CGGGGGGTCT GCGTCCACGG TAAAGACCCC
6481 GGGCAGCAGG CGCGCGTCGA AGTAGTCTAT CTTGCATCCT TGCAAGTCTA GCGCTGCTG
6541 CCATGCCGCG CGCGCAAGCG CGCGCTCGTA TGGGTGAGT GGGGGACCCC ATGGCATGGG
6601 GTGGGTGAGG GCGGAGGCGT ACATGCCGCA AATGTCGTAA ACGTAGAGGG GCTCTCTGAG
6661 TATTCCTAAG TATGTAGGGT AGCATCTTCC ACCGCGGATG CTGGCGCGCA CGTAATCGTA
6721 TAGTTCTGTC GAGGGAGCGA GGAGTCCGG ACCGAGGTTG CTACGGGCGG GCTGCTCTGC
6781 TCGGAAGACT ATCTGCTTGA AGATGGCATG TGAGTTGGAT GATATGGTTG GACGCTGAAA
6841 GACGTTGAAG CTGGCGCTCG TGAGACCTAC CGGCTCACGC ACGAAGGAGG CGTAGGAGTC
6901 GCGCAGCTTG TTGACCAGCT CGCGGTGAC CTGCACGTCT AGGGCGCAGT AGTCCAGGGT
6961 TTCTCTGATG ATGTCACTAT TATCTGTCC CTTTTTTTTC CACAGCTCGC GGTGAGGAC
7021 AAATCTTCCG CGGTCTTCC AGTACTCTTG GATCGGAAC CGTCCGGCCT CGGAACGGTA
7081 AGAGCCTAGC ATGTAGAACT GGTGACGCG CTGGTAGGCG CAGCATCCTT TTCTACGGG
7141 TAGCGCGTAT GCTTCCGCGG CCTTCCGAG CGAGGTGTTG GTGAGCGCAA AGGTGTCTCT
7201 GACCATGACT TTGAGTACT GGTATTTGAA GTCACTGTTC TCGCATCCCG CCTGCTCCCA
7261 GAGCAAAAAG TCCGTGCGCT TTTTGGAAAG CGGATTTGGC AGGGCGAAGG TGACATCGTT
7321 GAAGAGTATC TTTCCGCGCG GAGGCATAAA GTTGCCTGTG ATGCGGAAG GTCCCGGCAC
7381 CTGCGAAGCG TTGTTAAATTA CCTGGGCGCG GAGCAGATC TCGTCAAAGC CGTTGATGTT
7441 GTGGCCACCA ATGTAAAGTT CCAAGAAGCG CGGGATGCC TTGATGGAAG GCAATTTTTT
7501 AAGTTCCTCG TAGGTGAGCT CTTAGGGGA GCTGAGCGCG TGCTCTGAAA GGGCCAGTC
7561 TGCAAGATGA GGGTTGGAAG CGACGAATGA GCTCCACAGG TCACGGGCCA TTAGCATTTG
7621 CAGGTGGTCT CGAAAGGTCC TAACTGGCG ACCTATGGCC ATTTTTCTCT GGTGATGCA
7681 GTAGAAGGTA AGCGGCTCTT GTTCCAGCG GTCCCATCCA AGGTTCCGGG CTAGGCTCTC
7741 CGCGGCGATC ACTAGAGGCT CATCTCCGCC GAACCTCATG ACCAGCATGA AGGGCACGAG
7801 CTGCTTCCCA AAGGCCCCCA TCCAAGTATA GGTCTCTACA TCGTAGGTGA CAAAGAGACG
7861 CTGCTGCGA GGATGCGAGC CGATCGGGA GAACTGGATC TCCCGCCACC AATTGGAGGA
7921 GTGGCTATTG ATGTGTTGAA AGTAGAAGTC CCTGCGACGG GCCGAACACT CGTGTGCGT
7981 TTTGTAAAAA CGTGGCAGT ACTGGCAGCG GTGCACGGCG TGTACATCCT GCACGAGGTT
8041 GACCTGACGA CCGCGCACAA GGAAGCAGAG TGGGAATTTG AGCCCTCTGC CTGGCGGGT
8101 TGGCTGGTGG TCTTCTACTT CGGCTGCTTG TCCTTGACCG TCTGGCTGCT CGAGGGGAGT
8161 TACGTTGGAT CGGACCACCA CGCGCGGCGA GCCCAAAGTC CAGATGTCG CGCGCGCGCG
8221 TCGGAGCTTG ATGACAAATC CGCGCAGATG GGAGCTGTCC ATGGTCTGGA GCTCCCGCGG
8281 CGTCAGGTCA GCGGGGAGCT CCTGCAGGTT TACCTCGCAT AGACGGGTCA GGGCGGGGCG
8341 TAGATCCAGG TGATACCTAA TTTCCAGGGG CTGGTTGGTG GCGCGTCCA TGGCTTGCAA
8401 GAGGCCCGCAT CCGCGCGCGC CGACTACGCT ACCGCGCGCG GGGCGGTGG CCGCGGGGGT
8461 GTCTTGGATG GATGCATCTA AAGCGGTGA CGCGGGGAG CCGCGGAGG TAGGGGGGCG
8521 TCCGAGCCCG CCGGAGAGG GGGCAGGGCG ACGTCCGCGC CGCGCGCGG CAGGAGCTGG
8581 TGCTGCGCGC GTAGGTTGCT GCGGAACGCG ACGACGCGCG GGTGATCTC CTGAATCTGG
8641 CGCTCTGCGC TGAAGACGAC GGGCCCGGTG AGCTTGAGCC TGAAAGAGAG TTCACAGAA
8701 TCAATTTCCG TGTCGTTGAC GGCGGCTTGG CGCAAAATCT CTTGCACTG TCTGAGTTG
8761 TCTTGATAGG CGATCTCGGC CATGAACTGC TCGATCTCTT CCTCTGGAG ATCTCGCGT
8821 CCGGCTCGCT CCACGGTGGC GCGGAGGTGG TTGGAATGCG GGGCATGAG CTGCGAGAAG
8881 GCGTGAAGCG CTCCCTCGTT CCAGACGCGG CTGTAGACCA CGCCCCCTTC GGCATCGCGG
8941 GCGCGCATGA CCACCTGCGC GAGATTGAGC TCCACGTGCC GGGGGAAGAC GCGTAGTTT
9001 CGCAGGCGCT GAAAGAGGTA GTTGAGGGTG GTGCGGTGT GTTCTGCCAC GAAGAAGTAC
9061 ATAACCCAGC GTGCAACGCT GGATTCGTTG ATATCCCCCA AGGCTCAAG GCGCTCCATG
9121 GCCTCGTAGA AGTCCACGCG GAAGTTGAAA AACTGGAGT TGCGCGCGA CACGGTTAAC
9181 TCTCTCTCCA GAAGACGGAT GAGCTCGGCG ACAGTGTGCG GCACCTCGCG CTCGAAGGCT
9241 ACAGGGGCTT CTCTCTCTTC TTCAATCTCC TCTTCCATAA GGGCTTCCCC TTCTTCTCT

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FIGURE 22
(SHEET 3)

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3

9301 TCTGGCGGCG GTGGGGGAGG GGGGACACGG CGGCGACGAC GCGGCACCGG GAGGCGGTCTG
9361 ACAAGCGCT CGATCATCTC CCCGCGGCGA CGGCGCATGG TCTCGGTGAC GCGCGGGCCG
9421 TTCTCGCGGG GCGCGAGTTG GAAGACGCGG CCCGTCTAGT CCGGTTATG GGTTCGCGGG
9481 GGGCTGCCAT GCGGCAGGGA TACGGCGCTA ACAGTGCATC TCACCAATTG TTGTGTAGGT
9541 ACTCCGCGCG CGAGGGACCT GAGCGAGTCC GCATCGACCG GATCGSAAA CCTCTCGAGA
9601 AAGGCGTCTA ACCAGTCACA GTCGCAAGGT AGGCTGAGCA CCGTGGCGGG CGGCGAGCGGG
9661 CGGCGGTCCG GGTGTTTCT GCGGAGGTG CTGCTGATGA TGTAAITAAA GTAGGCGGTC
9721 TTGAGACGGC GATGTGTCGA CAGAAACACC ATGTCTTGG GTCCGGCTTG CTGAATGCGC
9781 AGGCGGTCCG CCATGCCCCA GGCCTCGTTT TGACATCGGC GCAGGTCTTT GTAGTAGTCT
9841 TGCAATGAGC TTTCTACCGG CACTTCTTCT TCTCTTCTCT CTGTCTCTGC ATCTCTTGCA
9901 TCTATCGCTG CGGCGGCGGC GAGTCTTGGC CGTAGGTGGC GCGCTCTTCC TCCCATGCGT
9961 GTGACCCCGA AGCCCCCAT CGGCTGAAGC AGGCTAGGT CCGCGAACAC GCGCTCGGCT
10021 AATATGCGCT GCTGCACCTG CGTGAAGGTA GACTGGAAGT CATCCATGTC CACAAAGCGG
10081 TGGTATCGCG CCGTGTGAT GGTGTAAGT CAGTTGGCCA TAACGGACCA GTTAACGGTC
10141 TGGTACCGCG GCTGCGAGAG CTCGCTGTAC CTCAGACGCG AGTAAGCCCT CGAGTCAAAT
10201 ACCTAGTCGT TGCAAGTCCG CACCAGGTAC TGGTATCCCA CCAAAAAGTG CGGCGGCGGC
10261 TGGCGGTAGA GCGGCCAGCG TAGGGTGGCC GGGGCTCCGG GGGCGAGATC TTCACACATA
10321 AGGCGATGAT ATCCGTAGAT GTACCTGGAC ATCCAGTGA TCCCGCGGC GGTGTGAGG
10381 GCGCGCGGAA AGTCGCGGAC CGGTTCCAG ATGTTGCGCA GCGGCAAAA GTGCTCCATG
10441 GTCCGGACGC TCTGGCGGT CAGGCGCGCG CAATCGTTGA CGCTCTAGCG TGCAAAAAGGA
10501 GAGCCTGTAA GCGGGCACTC TTCCGTGGTC TGGTGGATAA ATTGCAAGG GTATCATGGC
10561 GGACGACCGG GGTTCGAGCC CCGTATCCGG CCGTCCGCGG TGATCCATGC GGTTCACGCC
10621 CGCGTGTGCA ACCCAGGTGT GCGACGTGAG ACAACGGGGG AGTGCTCCTT TTGGCTTCTT
10681 TCCAGGCGCG GCGGCTGCTG CGCTAGCTTT TTGGGCACT GCGCGCGCG AGCGTAAGCG
10741 GTTAGGCTGG AAAGCGAAAG CATTAGTGG CTCGCTCCCT GTAGCGGAG GGTATTATTTC
10801 CAAGGCTTGA GTGCGGGGAC CCCGGTTGCG AGTCTCGGAC CCGCGGACT GCGCGGAAAG
10861 GGGGTTTGCC TCCCGCTCAT GCAAGACCCC GCTTGCATA TCCCTCGGAA ACAGGGACGA
10921 GCGGCTTTT TGTCTTTTCC AGATGCTCC GGTGCTGCGG CAGATGCGCC CCGCTCTCA
10981 GCAGCGGCAA GAGCAAGAGC AGCGGCGAGC ATGCAAGGCA CCGTCCCTCT CTCCTACCGC
11041 GTCAAGGAGG GCGACATCCG CGGTTGACCG GCGAGCAGAT GGTGATTACG AACCCCGCGG
11101 GCGCGCGGCC CGGCACTACC TGGACTTGGA GGAGGGCGAG GCGCTGGCGG GGTAGGAGC
11161 GCGCTCTCTT GAGCGGTACC CAAGGCTGCA GCTGAAGCGT GATACGCGTG AGGCGTACGT
11221 GCGCGCGCAG AACCTGTTTC GCGACCGGAG GGGAGAGGAG CCCGAGGAGA TGCGGATCG
11281 AAAGTCCAC GCAGGGCGCG AGCTGCGGCA TGGCTGTAAT CCGAGCGGT TCTGCGCGA
11341 GGAGGACTTT GAGCCCGACG CGCGAACCGG GATTAGTCCC GCGCGCGCAC ACGTGGCGGC
11401 CCGCGACCTG GTAAACCGAT ACGAGCAGAC GGTGAACCA GAGATTAACT TTCAAAAAG
11461 CTTTAAACAC CACGTGCGTA CGCTTGTTGG CCGCGAGGAG GTGGCTATAG GACTGATGCA
11521 TCTGTGGGAC TTTGTAAGCG CGCTGGAGCA AAACCAAAAT AGCAAGCCCG TCATGGCGCA
11581 GCTGTCTCTT ATAGTGCAGC ACAGCAGGSA CAACGAGGCA TTCAGGGATG CGCTGCTAAA
11641 CATAGTAGAG CCCGAGGGCC GCTGGCTGCT CGATTGATA AACATCTGCG AGAGCATAGT
11701 GGTGACGAGG CGCAGCTTGA GCTTGGCTGA CAAGGTGGCC GCCATCAACT ATTCATGCT
11761 TAGCCTGGGC AAGTTTACG CCGCAAGAT ATACCATACC CCTTACGTTT CCATAGACAA
11821 GGAGGTAAG ATCGAGGGGT TCTACATGCG CATGGCGCTG AAGGTGCTTA CTTGAGCGA
11881 CGACCTGGGC GTTTATCGCA ACGAGCGCAT CCACAGGCC GTGAGCGTGA GCGCGGCGG
11941 CGAGCTCAGC GACCGCGAGC TGATGCRAG CCGCAAGGCG GCGCTGGCTG GCACGGGCG
12001 CGCGATAGA GAGGCGGAGT CTTACTTTGA CGCGGGCGCT GACCTGCGCT GCGCCCAAG
12061 CCGACGCGCC CTGGAGGCGG ACCTGGGCTG CGGTTGGCAC CCGCGCGCGC CAGAGGACG
12121 TGGCAACGTC GCGCGCGTGG AGGAATATGA CGAGGACGAT GAGTACGAGC CAGAGGACG
12181 TGAGTACTAA CCGGTGATGT TTCTGATCAG ATGATGCAAG ACGCAACGGA CCGCGCGGTG
12241 CCGCGCGCGC TGCAAGGACA GCGTCCGCGC CTTAACTCCA CGGACGACTG GCGCGAGGTC
12301 ATGGACCGCA TCATGTGCTG GACTGCGCGC AATCTGACG CGTTCGGGCA GCAGCGCGC
12361 GCCAACCGCG TCTCCGCAAT TCTGGAAGCG GTGGTCCCGG CCGCGCGCAA CCGCACGCGC
12421 GAGAAAGTGC TGGCGATCGT AAACGCGCTG GCGGAAAACA GGGCATCCG GCGCGAGCG
12481 CCGCGCTGG TCTACGAGCG GCTGCTTCA GCGTGGCTC GTTACACAG CCGCAACGTG
12541 CAGACCAACC TGGACCGGCT GGTGGGGAT GTGCGGAGG CCGTGGCGCA GCGTACGCG
12601 GCGCAGCAG AGGGCAACCT GGGCTCCATG GTTGCACTAA ACGCTTCTT GAGTACACG
12661 CCGCGCAACG TGCGCGGGG ACAGGAGGAC TACACCACT TTGTGAGCGC ACTGCGGCTA

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FIGURE 22
(SHEET 4)

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12721 ATGGTGACTG AGACACCGCA AAGTGAGGTG TACCAGTCTG GGCCAGACTA TTTTTCAG
12781 ACCAGTAGAC AAGGCTTGCA GACCGTAAAC CTGAGCCAGG CTTTCAAAA CTTCAGGGG
12841 CTGTGGGGGG TGCGGGCTCC CACAGGCGAC CGCGGACCG TGCTAGCTT GCTGACGCCC
12901 AACTCGCGCC TGTGCTGCT GCTAATAGCG CCCTTCACGG ACAGTGGCAG CGTGCCCGG
12961 GACACATACC TAGGTCACCT GCTGACACTG TACCGCGAGG CCATAGGTCA GGCGCATGTG
13021 GACGAGCATA CTTTCCAGGA GATTACAAGT GTCAGCCGGG CGCTGGGGCA GGAGGACAG
13081 GGCAGCTGG AGGCAACCTT AAACCTACCTG CTGACCAACC GGCGGCAGAA GATCCCTCG
13141 TTGCACAGTT TAAACAGCGA GGAGGAGCGC ATTTTGCGCT ACCTGACGCA GAGCGTGAGC
13201 CTTAACCTGA TGCGGACGGG GGTAAACGCC AGCGTGGCGG TGGACATGAC CGCGGCAAC
13261 ATGGAACCGG GCATGTATGC CTCAAACCGG CCGTTTATCA ACCGCCATAA GGACTACTTG
13321 CATCGCGCGG CCGCGGTGAA CCCCAGTAT TTCAACCAATG CCATCTTGAA CCGCACTGG
13381 CTACCGCCCC CTGGTTTCTA CACCGGGGA TTCCAGGTGC CCGAGGTAA CGATGGATTG
13441 CTCTGGGACG ACATAGACGA CAGCGTGTIT TCCCGCAAC CGCAGACCTT GCTAGAGTTG
13501 CAACACGCGG AGCAGGCGA GGCGGCGCTG CGAAAGGAAA GCTTCCGCGA GCCAAGCAGC
13561 TTGTCCGATC TAGGCGCTGC GGCGCGCGG TCAGATGCTA GTAGCCCAT TCCAAGCTTG
13621 ATAGGGTCTC TTACCAGCAC TCGCACCAAC CGCGCGCGG TGCTGGGCGA GGAGGAGTAC
13681 CTAACNACT CGCTGCTGCA GCGCGACGCG GAAAAAACC TGCTCCGCG ATTTCCCAAC
13741 AACGGGATAG AGAGCCTAGT GGACAAGATG AGTAGATGGA AGACGTACGC GCAGGAGCAC
13801 AGGAGCGTGC CAGGCCCGCG CCGCGCCACC CGTCTCAAA GGCACGACCG TCAGCGGGGT
13861 CTGCTGTGGG AGGACGATGA CTCGGCAGAC GACAGCAGCG TCCTGGATT TGGAGGGAGT
13921 GGCAACCCGT TTGCGCACCT TCGCCCCAGG CTGGGGAGAA TGTTTTAAAA AAAAAAAGC
13981 ATGATGCAAA ATAAAAAAT CACCAAGGCC ATGGCACCAG CCGTTGGTT TCTTGTATTG
14041 CCCTTAGTAT GCGCGCGCG GCGATGTATG AGGAAGTCC TCCTCCCTCC TACGAGAGTG
14101 TGGTGAGCGC GCGCGCAGTG GCGCGCGCG TGGGTTCTCC CTTCGATGCT CCGCTGGACC
14161 CGCGGTTTGT GCCTCCGCGG TACCTGCGG CTACCGGGG GAGAAACAGC ATCCGTTACT
14221 CTGAGTTGGC ACCCTATTTC GACACCAACC GTGTGTACCT GGTGGCAAC AAGTCAACGG
14281 ATGTGGCATC CCTGAATAC CAGAACGACC ACAGCAACT TCTGACCAG GTCAATCAAA
14341 ACATGACTA CAGCCCGGGG GAGGCAAGCA CACAGACCT CAATCTTGAC GACCGGTGCG
14401 ATCGGGGCGG CGACCTGAAA ACCATCTGTC ATACCAACAT GCCAAATGTG AACGAGTTCA
14461 TGTTTACCAA TAACTTTAAG GCGGGGTGA TGGTGTGCG CTTCCTACT AAGGACAATC
14521 AGGTGAGCTG GAAATACGAG TGGGTGGAGT TCACGTGCC CGAGGGCAAC TACTCCGAGA
14581 CCATGACCAT AGACCTTATG AACAAACGCA TCGTGGAGCA CTACTTGAAA GTGGGAGAGC
14641 AGAACGGGTG TCTGGAAGC GACATCGGG TAAAGTTTGA CACCCGCAAC TTCAGACTGG
14701 GGTTTGACCC CGTCACTGGT CTGTGATGCT CTGGGGTATA TACAAACGAA GCCTTCCATC
14761 CAGACATCAT TTTGCTGCCA GGAATCGGGG TGGACTTCAC CCACAGCCG CTGAGCAACT
14821 TGTGGGCAT CCGCAAGCG CAACCTTCC AGGAGGCTT TAGGATCACC TACGATGATC
14881 TGGAGGGTGG TAACATTCCC GCACTGTTGG ATGTGGACGC CTACCAGGCG AGCTTGAAG
14941 ATGACACCGA ACAGGGCGGG GGTGGCGCAG CGGCGAGCAA CAGCAGTGGC AGCGGCGCGG
15001 AAGAGAACTC CAACCGCGCA GCGCGGCAA TGCAGCCGCT GGAGGACATG AACGATCATG
15061 CCATTGCGCG CGACACCTTT GCCACACGG CTGAGGAGAA GCGCGCTGAG GCGGAAGCAG
15121 CGGCGGAAGC TGCCGCCCC GCTGCGCAAC CCGAGGTGGA GAAGCCTCAG AAGCAACCGG
15181 TGATCAAAAC CCTGACAGAG GACAGCAAGA AACGCAGTTA CAACCTAATA AGCAATGACA
15241 GCACCTTCAC CCACTACCGC AGCTGGTACC TTGCATACAA CTACGGCGAC CCTCAGACCG
15301 GAATCGCTC ATGGACCTG CTTTGCACTC CTGAGTAAAC CTGCGGCTCG GAGCAGGTCT
15361 ACTGGTGGT GCCAGCATG ATGCAAGACC CGTGACCTT CCGCTCCAGC GCGCAGATCA
15421 GCAACTTTCC GGTGGTGGG GCGGAGCTGT TGCCGTTGCA TTCCAAGAGC TTCTACAACG
15481 ACCAGGCGGT CTACTCCCAA CTCATCCGCC AGTTTACCTC TCTGACCCAC GTGTTCAATC
15541 GCTTTCCCGA GAACAGATT TTGGGCGGCC CGCCAGCCCC CACCATCACC ACCGTGAGT
15601 AAAACGTTC TGCTCTACA GATCACGGGA CGCTACCGCT CCGCTACGTT TACAGGGCC
15661 TCCAGCGAGT GACCATTAAT GACGCCAGAC GCGCACCTG CCGCTACGTT TACAGGGCC
15721 TGGGCATAGT CTGCGCGCG GTCCTATCGA GCGCACTTT TTGAGCAAGC ATGTCCATCC
15781 TTATATCGCC CAGCAATAAC ACAGGCTGGG GCGTGGCTT CCAAGCAAG ATGTTTGGCG
15841 GGGCCAGAA GCGCTCCGAC CAACACCCAG TGCGGTGCG CGGCGACTAC CCGCGCGCCT
15901 GGGGCGCGCA CAAACGCGGC CGCACTGGG GCACCAACGT CGATGACGCC ATCGAGCGGG
15961 TGTGGAGGA GCGCGCGAAC TACAGGCCA CCGCGCCACC AGTGTCCACA GTGAGCGCGG
16021 CCATTAGAC CGTGGTGGC GGAGCCCGCG GCTATGCTAA AATGAAGAGA CCGCGAGGCG
16081 GCGTAGCAGC TCGCCACCGC CCGCGACCG GCACTGCGCG CCAACGCGCG GCGCGCGCGG

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FIGURE 22
(SHEET 5)

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16141 TGCTTAACCG CGCAGCTCGC ACCGGCCGAC GGGCGGCCAT GCGGGCCGCT CGAAGGCTGG
 16201 CCGCGGGTAT TGTCACTGTG CCCCCCAGGT CCAGGCGACG AGCGGCCGCC GCAGCAGCCG
 16261 CGGCCATTAG TGCTATGACT CAGGGTGGCA GGGGCAACGT GTATTGGGTG CGCGACTCGG
 16321 TTAGCGGCGCT GCGCGTGCCC GTGGCACCAC GCGCCCGCGG CAAGTAGATT GCAAGAAAAA
 16381 ACTACTTAGA CTCGTACTGT TGTATGTATC CAGCGCGCGC GCGCGCGAAC GAAGCTATGT
 16441 CCAAGCGCAA AATCAAAGAA GAGATGCTCC AGGTCACTGC GCGCGAGATC TATGGCCCCC
 16501 CGAAGAAGGA AGAGCAGGAT TACAAGCCCC GAAAGCTAAA GCGGGTCAAA AAGAAAAAGA
 16561 AAGATGATGA TGTGAACCTT GACGACGAGG TGGAACTGCT GCACGCTACC GCGCCGAGGC
 16621 GACGGGTACA GTGGAAAGGT CGACGCGTAA AACGTGTTTT GCGACCCGCG ACCACCGTAG
 16681 TCTTTACGCC CGGTGAGGCG TCCACCAGCA CCTACAAGCG CGTGTATGAT GAGGTGTACG
 16741 GCGACGAGGA CCTGCTTGAG CAGGCCAACG AGCGCCTCGG GGAGTTTGCC TACGGAAAGC
 16801 GGCATAAGGA CATGCTGGCG TTGCCGCTGG ACGAGGGCAA CCCAACACCT AGCCTAAAGC
 16861 CCGTAACACT GCAGCAGGTG CTGCCCGCGC TTGCACCGTC CGAAGAAAAA GCGCGCTTAA
 16921 AGCGCGAGGT TGGTGACTTG GCACCCACCG TGCACTGAT GGTACCCAGG GCGCAGCGAC
 16981 TGGAAAGTGT CTGGAAAAAT ATGACCGTGG AACCTGGGCT GGAGCCGAG GTCCCGGTGC
 17041 GGCCAATCAA GCAGGTGGCG CCGGACTGG GCGTGACGAC CGTGGACGTT CAGATACCCA
 17101 CTACCACTAG CACCACTATT GCCACCGCCA CAGAGGGCAT GGAGACACAA ACGTCCCCGG
 17161 TTGCTCTAGC GGTGGCGGAT GCGCGGCTGC AGCGCTCGC TGCGGCCGCG TCCAAGACCT
 17221 CTACGGAGGT GCAAAACGAC CCGTGGATGT TTCGCGTTTC AGCCCCCGCG CGCCCGCGCG
 17281 GTTCGAGGAA GTACGGCGCC GCCAGCGCGC TACTGCGCGA ATATGCGCTA CATCTCTCCA
 17341 TTGCGCCTAC CCGCGCTAT CCGCGCTACA CCTACCGCCC CAGAAGACGA GCAACTACCC
 17401 GAGCGCGAAC CACCACTGGA ACCCGCGCCG GCGGTGCGCG TCGCCAGCCC GTGCTGGGCC
 17461 CGATTTCGGT GCGCAGGTG GCTGCGAAG GAGGCAGGAC CCTGGTGTCT CCAACAGCGC
 17521 GCTACCAACC CAGCATCGTT TAAAGCCGCG TCTTTGTGGT TCTTGCAGAT ATGGCCCTCA
 17581 CCGTCCGCGT CCGTTTCCCG GTGCCCGGAT TCCGAGGAAG AATGCACCGT AGGAGGGGCA
 17641 TGGCGCGCCA CCGCTGACG GCGCGCATGC GTCGTGCGCA CCACCGCGCG CGCGCGCGCT
 17701 CGCACCGTCG CATGCGCGCG GGTATCTCGT CCTCTCTTAT TCCACTGATC GCGCGCGCGA
 17761 TTGGCGCGGT GCGCGGAATT GCATCCGTGG CCTTGCAGGC GCAGAGACAC TGATTAAAAA
 17821 CAAGTTGATG GTGGAATAAT CAAATAAATA AGTCTGGACT CTCACGCTCG CTGTGTCTCT
 17881 TAACTATTTT GTAGAATGGA AGACATCAAC TTTGCGTCTC TGGCCCGCGG ACACGGCTCG
 17941 CGCCCGTTCA TGGGAAACTG GCAAGATATC GGCACACGA ATATGAGCGG TGGCGCCTTC
 18001 AGCTGGGGCT CGCTGTGGAG CGGCATTAAA AATTTCCGTT CCACCGTTAA GAACTATGGC
 18061 AGCAAGGCCCT GGAACAGCAG CACAGGCCAG ATGCTAGGGG ATRAGTTGAA AGAGCAAAAT
 18121 TTCCAACAAA AGGTGGTAGA TGGCCTGGCC TCTGGCATTG GCGGGGTGGT GGACCTGGCC
 18181 AACCAGGCAG TGCAAAATAA GATTAAACAGT AAGCTTGATC CCGCCCTTCC CGTAGAGGAG
 18241 CCTCCACCGG CCGTGGAGAC AGTGTCTCCA GAGGGGCGTG GCGAAAAGCG TCCGCGCCCC
 18301 GACAGGGGAG AAACCTCTGGT GACGCAATAA GACGAGCCTC CCTCGTACGA GGAGGCACTA
 18361 AAGCAAGGCC TGCCCAACCAC CCGTCCCATC GCGCCCATGG CTACCGGAGT GCTGGGCCAG
 18421 CACACACCGG TAACGCTGGA CCGTCCCTCC CCGCGCGACA CCCAGCAGAA ACGTGTGCTG
 18481 CCAGGCCCGA CCGCGTTGT TGTAAACCGT CCTAGCGCGG CGTCCCTGCG CCGCGCGCGC
 18541 AGCGGTCCGC GATCCTTTCG GCGCGTAGCC AGTGGCAACT GGCAAAAGCA ACTGAACAGC
 18601 ATCGTGGGTC TGGGGGTGCA ATCCCTGAAG CGCCGACGAT GCTTCTGAAT AGCTAACGTC
 18661 TCGTATGTGT GTCAATGATG CGTCCATGTC GCGCCAGAG GAGCTGCTGA GCGCGCGCGC
 18721 CCGCGCTTTC CAAGATGGCT ACCCTTTCGA TGATGCGGCA GTGGTCTTAC ATGCACATCT
 18781 CCGGCCAGGA CGCCTCGGAG TACCTGAGCC CCGGGCTGGT GCGAGTTTGC CCGCCACCGG
 18841 AGACGTACTT CAGCCTGAAT AACAAGTTTA GAAACCCAC GGTGGCGCCT ACGCACGACG
 18901 TGACCAACGA CCGTCCCGAG CGTTTGACGC TGCGGTTTAT CCCTGTGGAC CGTGAGGATA
 18961 CTGCGTACTC GTACAAGCGG CCGTTCACCC TAGCTGTGGG TGATAACCGT GTGCTGGACA
 19021 TGGCTTCCAC GTACTTTGAC ATCCGCGCGG TGCTGGACAG GGGCCCTACT TTTAAGCCCT
 19081 ACTCTGGCAC TCCCTACAAC GCCCTGGCTC CCAAGGGTGC CCCAAATCCT TGCGAATGGG
 19141 ATGAAGCTGC TACTGCTCTT GAAATAAACC TAGAAGAAGA GGACGATGAC AAGCAAGACG
 19201 AAGTAGACGA GCAAGCTGAG CAGCAAAAAA CTCACGTATT TGGCGAGCGG CCTTATCTCG
 19261 GTATAAATAT TACAAGGAG GGTATTCAAA TAGGTGTGCA AGGTCAAAAC CCTAATATG
 19321 CCGATAAAAC ATTTCAACCT GAACCTCAAA TAGGAGATC TCAGTGTGAC GAACTGAAA
 19381 TTAATCATGC AGCTGGGAGA GTCCCTAAAA AGACTACCCC AATGAACCA TGTACCGGT
 19441 CATATGCAAA ACCCACAAAT GAAAATGGAG GGCAAGGCAT TCTTGTAAAG CAACAAAATG
 19501 GAAAGCTAGA AAGTCAAGTG GAAATGCAAT TTTTCTCAAC TACTAGAGCG ACCGACGGCA

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 FIGURE 22
 (SHEET 6)

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19561 ATGGTGATAA CTTGACTCCT AAAGTGGTAT TGTACAGTGA AGATGTAGAT ATAGAAACCC
19621 CAGACACTCA TATTTCTTAC ATGCCCACTA TTAAGGAAGG TAACACAGA GAACATATGG
19681 GCCAACAAATC TATGCCCAAC AGGCCCTAAT ACATTGCTTT TAGGGACAAT TTTATTGGTC
19741 TAATGTATTA CAACAGCACG GGTAAATATG GTGTTCTGGC GGGCCAAGCA TCGCAGTTGA
19801 ATGCTGTTGT AGATTTGCAA GACAGAAACA CAGAGCTTTC ATACCAGCTT TTGCTTGATT
19861 CCATTGGTGA TAGAACCAGG TACTTTTCTA TGTGGAATCA GGCTGTTGAC AGCTATGATC
19921 CAGATGTTAG AATTATTGAA AATCATGGAA CTGAAGATGA ACTTCCAAAT TACTGCTTTC
19981 CACTGGGAGG TGTGATTAAAT ACAGAGACTC TTACCAAGGT AAAACCTAAA ACAGGTCAGG
20041 AAAATGGATG GAAAAAAGAT GCTACAGAAT TTTCCAGATA AAATGAAATA AGAGTTGSA
20101 ATAATTTTGC CATGGAAATC AATCTAAATG CCAACCTGTG GAGAAATTC CTGTACTCCA
20161 ACATAGCGCT GTATTTGCCG GACAAGCTAA AGTACAGTCC TTCCAACGTA AAAATTTCTG
20221 ATACCCCAA CACCTACGAC TACATGAACA AGCGAGTGGT GGCTCCCGGG TTAGTGGACT
20281 GCTACATTAA CTTGGAGCA CGCTGGTCCC TTGACTATAT GACAAACGTC AACCCATTTA
20341 ACCACCACCG CAATGCTGGC CTGCGCTACC GCTCAATGTT GCTGGGCAAT GGTGCTATG
20401 TGCCCTTCCA CATCCAGGTG CCTCAGAAGT TCTTTGCCAT TAAAAACCTC CTCTCCTG
20461 CGGGCTCATA CACCTACGAG TGGAACTTCA GGAAGGATGT TAACATGTTT CTGCAGAGCT
20521 CCTAGGAAA TGACCTAAGG GTTGACGGAG CCAGCATTAA GTTTGATAGC ATTTGCTTTT
20581 ACGCCACCTT CTTCCCATG GCCACAACA CGCCCTCCAC GCTTGAGGCC ATGCTTAGAA
20641 ACAGACCAA CGACAGTCC TTTAAGACT ATCTCTCCG CGCCAACATG CTCTACCTTA
20701 TACCCGCAA CGCTACCAAC GTGCCATAT CCATCCCTC CGCAACTGG CGGGCTTCC
20761 GCGGCTGGC CTTCACGCG CTTAAGACTA AGGAAACCCC ATCACTGGG TCGGGCTAGC
20821 ACCCTTATTA CACCTACTCT GGCTCTATAC CCTACCTAGA TGGAACTTTT TACCTCAAC
20881 ACACCTTTAA GAAGTGGGCC ATTACCTTTG ACTCTTCTGT CAGCTGGCTT GGCATGACC
20941 GCCTGCTTAC CCCCAACGAG TTTGAAATTA AGCGCTCAGT TGACGGGAG GGTIACAAG
21001 TTGCCCACTG TAACATGACC AAAGACTGGT TCTTGGTACA AATGCTAGCT AACTACAACA
21061 TTGGCTACCA GGGCTTCTAT ATCCCAAGA GCTACAAGGA CCGCATGTAC TCCTTCTTTA
21121 GAAGCTTCCA GCCCATGAGC CGTCAGGTGG TGGATGATAC TAAATACAAG GACTACCAAC
21181 AGGTGGGCAT CCTACACCAA CACAACAAC CTGATTTGTT TGGCTACCTT GCCCCACCA
21241 TGCGGGAAGG ACAGGCCTAC CTGTGTAAC TCCCTATCC GCTTATAGGC AAGACCGCAG
21301 TTGACAGCAT TACCCAGAAA AAGTTTCTTT GCGATCGCAC CCTTTGGCG ATCCCATCT
21361 CCAAGTAAC TTATGTCATG GCGCACTCA CAGACCTGGG CCAAAACCTT CTCTACGCCA
21421 ACTCCGCCCA CGCGTAGAC ATGACTTTTG AGGTGGATCC CATGGACGAG CCCACCTTC
21481 TTTATGTTTT GTTTGAAGTC TTTGACGTGG TCGGTGTGCA CCGGCCGCAC CGCGCGTCA
21541 TCGAAACCGT GTACCTGCGC ACGCCCTTCT CGGCCGCAA CGCCACAACA TAAAGAAGCA
21601 AGCAACATCA ACAACAGCTG CGGCATGGG CTCCAGTAG CAGGAATGA AAGCCATTGT
21661 CAAGATCTT GGTGTGGGCT CATATTTTCT GGGCACCTAT GACAAGCGCT TTCCAGGCTT
21721 TGTTCCTCCA CACAAGCTCG CCTGGCCAT AGTCAATACG GCCGTGCGG AGACTGGGG
21781 CGTACACTGG ATGGCCTTTG CCTGGAACCC GCACCTCAAAA ACATGTCTAC TCTTTGAGCC
21841 CTTTGGCTTT TCTGACCAGC GACTCAAGCA GGTTTACCAG TTTGAGTACG AGTCACTCT
21901 GCGCGTAGC GCATTGCTT CTTCGCCGA CCGCTGTATA ACGCTGGAAG AGTCCACCCA
21961 AAGCGTACAG GGGCCCAACT CGGCCGCTG TGGACTATTC TGCTGCATGT TTCTCCACG
22021 CTTTGCCAA TGGCCCAA CTCCATGGA TCACAACCCC ACCATGAACC TTATTAACCG
22081 GGTACCCAA TCCATGCTCA ACGTCCCCA GGTACAGGCC ACCCTGCGTC GCAACAGGA
22141 ACAGCTCTAC AGCTTCTGAG AGCGCCACTC GGCCTACTTC CGCAGCCACA GTGCGCAGAT
22201 TAGGAGCGCC ACTTCTTTT GTCACTTGAA AAACATGTAA AAATAATGTA CTAGAGACAC
22261 TTTCAATAAA GGCAAATGCT TTTATTTGTA CACTCTGGG TGATTTATTA CCCCACCTC
22321 TGCGCTGTC GCGGTTTAAA AATCAAAGGG GTTCTGCGC GCATCGCTAT GCGCCACTGG
22381 CAGGGACACG TTGGATACT GGTGTTTGT GCTCCACTTA AACTCAGGCA CAACCATCCG
22441 CGCGAGCTCG GTGAAGTTT CACTCCACAG GCTGCGCACC ATCACCACG CGTITAGCAG
22501 GTCGGGCGCC GATATCTTGA AGTCGCAGTT GGGGCTCCG CCTGCGCGC GCGAGTTGG
22561 ATACACAGGG TTGCAGCACT GGAACACTAT CAGCGCCGG TCGTGCACGC TGGCCAGCAC
22621 GCTCTTGTG GAGATCAGAT CCGGTCAGG GTCTCCGCG TTGCTCAGGG CGAACGAGT
22681 CAACITTTGG AGCTGCCTTC CCAAAAAGGG CGCGTGCCCA GGCTTTGAGT TGCACTCGCA
22741 CCGTAGTGGC ATCAAAAGGT GACCGTGCCC GGTCTGGCG TTAGGATACA GCGCTGCAT
22801 AAAAGCTTTG ATCTGCTTAA AAGCCACCTG AGCCTTTGCG CTTTCAGAGA AGAACATGCC
22861 GCAAGACTTG CCGGAAACT GATTGGCCGG ACAGGCGCG TCGTGCACGC AGCACCTTGC
22921 GTCGGTGTG GAGATCTGCA CCACATTTG GCGCCACCG TTCTTACGA TCTTGGCCTT

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FIGURE 22
(SHEET 7)

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22981 GCTAGACTGC TCCTTCAGCG CGCGCTGCCG GTTTTCGCTC GTCACATCCA TTTCAATCAC
23041 GTGCTCCTTA TTTATCATAA TGCTTCGGTG TAGACACTTA AGCTCGCCTT CGATCTCAGC
23101 GCAGCGGTGC AGCCACAACG CGCAGCCCGT GGGCTCGTGA TGCTTGTAGG TCACCTCTGC
23161 AAACGACTGC AGGTACGGCT GCAGGAATCG CCCCATCATC GTCACAAAGG TCTTGTGCT
23221 GGTGAAGGTC AGCTGCAACC CGCGTGCTC CTGTTTCAGC CAGGTCTTGC ATACGGCGCG
23281 CAGAGCTTCC ACTTGGTCAG GCAGTAGTTT GAAGTTCGCC TTTAGATCGT TATCCACGTG
23341 GTACTTGTCC ATCAGCGCGC GCGCAGCCTC CATGCCCTTC TCCCACGCGC ACACGATCGG
23401 CACACTCAGC GGGTTTCATCA CCGTAATTC ACTTTCCGCT TCGCTGCGCT CTTCCTCTTC
23461 CTCTTGGTGC CGCATACCAC GCGCCACTGG GTGCTCTTCA TTCAGCCGCG GCACGTGTGG
23521 CTTACCTCCT TTGCCATGCT TGATTAGCAC CGGTGGGTTG CTGAAACCCA CCATTGTAG
23581 CGCCACATCT TCTCTTCTT CCTCGCTGTC CACGATTACC TCTGGTGATG GCGGGCGCTC
23641 GGGCTTGGGA GAAGGGCGCT TCTTTTCTT CTGCGCGCA ATGCCAAAT CCGCGCGCGA
23701 GGTGATGGC CGCGGGCTGG GTGTGCGCG CACCAGCGCG TCTTGTGATG AGTCTTCTC
23761 GTCTCTGGAC TCGATACGCC GCCTCATCGC CTTTTTGGG GCGCGCGCGG GAGGCGCGCG
23821 CGACGGGAC GGGGACGACA CGTCTCCAT GGTGGGGGA CGTCGCGCG CACGCGCTCC
23881 GCGCTCGGG GTGCTTTCG GCTGCTCCTC TTCCCGACTG GCCATTCTCT TCTCTATAG
23941 GCAGAAAAAG ATCATGGAGT CAGTCGAGAA GAAGGACAGC CTAACGCGCC CCTCTGAGTT
24001 CGCCACCCAC GCCTCCACCG ATGCCGCCAA CGCGCCTACC ACCTTCCCGC TCGAGGCACC
24061 CCCGCTTAG GAGGAGGAAG TGATTATCGA CGAGGAACCA GGTTTGTAA GCGAGACGA
24121 CGAGGACCGC TCAGTACCAA CAGAGGATAA AAAGCAAGAC CAGGACAAAG GCGAGACGA
24181 CGAGGAACAA GTCGGGCGGG GGGACGAAG GCATGGCGAC TACCTAGATG TGGGAGACGA
24241 CGTCTGTTG AAGCATCTGC AGCGCCAGTG CGCCATTATC TGCGACGCGT TGCAGAGCG
24301 CAGGATTTG CCCCTCGCCA TAGCGATGT CAGCCTTGCC TAOGAACGCC ACCTATTCTC
24361 ACCGCGGTA CCCGCCAAAC GCGCAGATGC GAGCCCAACC CGCGCTCAA
24421 CTCTACCCG GTATTTGCCG TGCCAGAGGT GCTTGCCACC TATCATCTCT TTTTCCAAA
24481 CTGCAAGATA CCCCTATCCT GCGGTGCCAA CGCGAGCCGA GCGGACAAAG AGCTGGCTT
24541 GCGGCGGGC GCTGTATAC CTGATATGCT CTGCTCAAC GAAGTGCCAA AATCTTTGA
24601 GGTCTTTGA CGCGACGAGA AGCGCGCGCG AAACGCTCTG CAACAGGAAA ACAGCGAAA
24661 TGAAGTCACT TCTGAGTGT TGTTGGAAC CTGAGGTGAC AACCGCGCC TAGCGTACT
24721 AAATTTGCAA GAACAAACAG AGGAGGCGCT ACCCGAGTT GCGACGAGC AGCTAGCGCG
24781 CATGAGCACA GTCATGAGTG AGCTGATCGT CGCGCGTGG GCGGACGAGC AGCTAGCGCG
24841 AAATTTGCAA GAACAAACAG AGGAGGCGCT ACCCGAGTT GCGACGAGC AGCTAGCGCG
24901 CTGGCTTCAA ACGCGCGAGC CTGCGACTTT GAGGAGGCGT TTTGCTGACC CGGAGATGCA
24961 AGTGTCTGTT ACCGTGGAGC TTGAGTGCAT GCAGCGGTTT TTTGCTGACC CGGAGATGCA
25021 GCGCAAGCTA GAGGAACAT TGCATACAC CTTCGACAG GGTCTACGAC GCGAGGCTG
25081 CAAGATCTCC AACGTGGAGC TCTGCAACCT GTTCTCTTAC CTGTGAATTT TGCAGGAAA
25141 CGCGCTTGGG CAAAACGTGC TTCAITCCAC GCTCAAGGCG GAGGCGCGCC GCGACTACGT
25201 CGCGACTGC GTTACTTAT TTCTATGCTA CACTGGCAG ACGGCCATGG GCGTTTGGCA
25261 GCAATGCTTG GAGGAGTGCA ACCTCAAGGA GCTGCAGAAA CTGCTAAGC AAAACTTGA
25321 GGACCTATGG ACGGCTTCA ACGAGCGCTC CGTGGCGCG CACTGGCGG ACATCATTTT
25381 CCCGAAACGC CTGCTTAAAA CCTGCAACA GGGTCTGCCA GACTTCACCA GTCAAAGCAT
25441 GTTGCAAGAC TTTAGGAAC TTATCCTAGA GCGCTCAGGA ATCTTGCCCG CCACCTGCTG
25501 TGCACTTCTC AGCGACTTTG TGCCATTAA GTACCGGAA TGCCCTCCCG CGCTTTGGGG
25561 CCACCTGCTAC CTCTGCGAGC TAGCCAACTA CCTTGCTTAC CACTCTGACA TAATGGAAGA
25621 CGTGAAGGCT GACGCTTAC TGGAGTGCA CTGTGCTGC AACCTATGCA CCCGCAACG
25681 CTCCCTGGTT TGCAATTCGC AGCTGCTTAA CGAAAGTCAA ATTATCGSTA CCTTTGAGCT
25741 GCAGGGTCCC TCGCTGACG AAAAGTCCGC GGTCCCGGG TTGAACTCA CTCGGGGCT
25801 GTGACGCTG GCTTACCTTC GCAATTTGT ACCTGAGGAC TACCACGCCC ACGAGATTAG
25861 GTTCTACGAA GACCAATCCC GCGCGCCAAA TGCGAGCTT ACCGCTTGGC TCATTACCCA
25921 GGGCCACAT CTGCGCAAT TGCAAGCCAT CAACAAAGCC CGCCAGAGT TTCTGTACG
25981 AAAGGACGG GGGTTTACT TGGACCCCA GTCCGCGAG GAGCTCAACC CAATCCCCC
26041 GCGCGCGCG CCTATCAGC AGCAGCGCG GGCCTTGTCT TCCAGGATG GCACCCAAA
26101 AGAAGCTGCA GCTGCGCGCG CACCCACGG ACGAGGAGGA ATACTGGGAC AGTCAGGCG
26161 AGGAGGTTT GAGCGAGGAG GAGGAGGACA TGATGGAAGA CTGGGAGAGC CTAGAGGAG
26221 AAGCTTCCGA GGTGGAAGAG GTGTGAGAG AAACACCGTC ACCCTCGGTC GCATTCCTCT
26281 CGCGCGCGC CCAGAAATCG GCAACCGTT CCAGCATGGC TACACCTCTC GCTCTCAGG
26341 CGCGCGCGC ACTGCCCGTT CGCGACCCA ACCGTAGATG GGACACCACT GGAACAGGG

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FIGURE 22
(SHEET 8)

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26401 CCGGTAAGTC CAAGCAGCGG CCGCCGTTAG CCCAAGAGCA ACAACAGCGC CAAGGCTACC
 26461 GCTCATGGCG CGGGCACAAG AACGCCATAG TTGCTTGCCTT GCAAGACTGT GGGGGCAACA
 26521 TCTCTTCGCG CCGCCGCTTT CTCTCTTACC ATCACGGCGT GGCCTTCCCC CGTAACATCC
 26581 TGCATTACTA CCGTCATCTC TACAGCCCAT ACTGCACCGG CGGCAGCGGC AGCGGCAGCA
 26641 ACAGCAGCGG CCACACAGAA GCAAAGGCGA CCGGATAGCA AGACTCTGAC AAAGCCCAAG
 26701 AAATCCACAG CCGCGGCAGC AGCAGGAGGA GGAGCGCTGC GTCTGGCGCC CAACGAACCC
 26761 GTATCGACCC GCGAGCTTAG AAACAGGATT TTTCCACTC TGTATGCTAT ATTTCAACAG
 26821 AGCAGGGGCC AAGAACAGAA GCTGAAATA AAAACAGGT CTCTGCGATC CCTCACCCGC
 26881 AGCTGCGTGT ATCACAAAG CGAAGATCAG CTTCGGCGCA CGCTGGAAGA CGCGAGGCT
 26941 CTCTTCAGTA AATACTGCGC GCTGACTCTT AAGGACTAGT TTCCGCGCCT TTCTCAAAAT
 27001 TAAGCGCGAA AACTACGTCA TCTCCAGCGG CCACACCCGG CGCCAGCACC TGTCGTCAGC
 27061 GCCATTATGA GCAAGGAAAT TCCACGCCCC TACATGTGGA GTTACCGACC ACAAAATGGA
 27121 CTTGCGGCTG GAGCTGCCCA AGACTACTCA ACCCGAATAA ACTACATGAG CGCGGAGCCC
 27181 CACATGATAT CCCGGGTCAA CGGAATCCGC GCCCACGAA ACCGAATCTCT CTGTGAACAG
 27241 CGCGCTATTA CCACCACACC TCGTAATAAC CTTAATCCCC GTAGTTGGCC CGCTGCCCTG
 27301 GTGTACCAAG AAAGTCCCCG TCCCACCACT GTGGTACTTC CCAGAGACGC CCAGGCGGAA
 27361 GTTCAGATGA CTAAGTCAGG GCGCGAGCTT CGCGGCGGCT TTGCTCACAG GGTGCGGTG
 27421 CCGCGGCGAG GTATAACTCA CCTGACAAAT AGAGGGCGAG GTATTGAGCT CAACGACGAG
 27481 TCGGTGAGCT CCTCGCTTGG TCTCGTCCG GACGGGACAT TTCAGATCGG CGGCGCGCG
 27541 CGTCTCTCAT TCACGCTCTG TCAGGCAATC CTAAGTCTGC AGACTCTGTC CTCTGAGCGG
 27601 CGCTCTGGAG GCATTGGAAC TCTGCAATTT ATTGAGGAGT TTGTGCCATG GGTCTACTTT
 27661 AACCCCTTCT CGGACCTTCC CGGCCACTAT CCGGATCAAT TTAATCTCAA CTTTGAGCGG
 27721 GTAAAGGACT CGGCGGACGG CTAAGACTGA TAATTAAGTG GAGAGGACGA GCAACTGCGC
 27781 CTGAAACACC TGGTCCACTG TCGCGCCAC AAGTGTCTTG CCGCGGACTC CGGTGAGTTT
 27841 TGCTACTTTG AATTGCCCCG GGATCATATC GAGGATCTTT GTTGCCATCT CTGTGCTGAG
 27901 TATAATAAAT ACAGAAATTA AAATATACTG GGGCTCTAT CGCCATCCTG TAAACGCCAC
 27961 CGTCTCTCAC CGCCCAAGCA AACCAAGGCG AACCTTACCT GGTACTTTTA ACATCTCTCC
 28021 CTCTGTGATT TACAACAGTT TCAACCCAGA CGGAGTGAGT CTACGAGAGA ACCTCTCGGA
 28081 GCTCAGCTAC TCCATCAGAA AAACACCCAC CCTCCTTACC TGCCGGGAAC GTACCTTAA
 28141 TTAAGATGCA GCCTTCTGCG ATGTACAGAT CTGACTTTGG CCAGCACCTG TCCCGCGGAT
 28201 TTGTCCAGT CCAACTACAG CGACCCACCC TAACAGAGAT GACCAACACA ACCAACGCGG
 28261 CCGCGCTAC CGGACTTACA TCTACCACAA ATACACCCCA AGTTTCTGCC TTGTCAATA
 28321 ACTGGGATAA CTTGGGCAAT TGGTGGTTCT CCATAGCGCT TATGTTTGTA TGCCTTATTA
 28381 TTATGTGGCT CATCTGCTGC CTAAGCGCA AACGCGCCCG ACCACCCATC TATAGTCCCA
 28441 TCATTGTGCT ACACCCAAAC AATGATGGA TCCATAGATT GGACGGAGTG AAACACATGT
 28501 TCTTTTCTCT TACAGTATGA TTAATGAGA TTAATTAAGG AATTTCTGTC CAGTTTATTC
 28561 AGCAGCACTC CTTGCCCCCT CTCCAGCTC TGGTATTGCA GCTTCCTCTT GGTGCGAAC
 28621 TTCTCCACA ATCTAAATGG AATGTCAGTT TCTCTCTGTT CTTGTCCTAT CGCACCCACT
 28681 ATCTTCATGT TGTTCAGAT GAAGCGGCGA AGACCGTCTG AAGTACCTT CAACCCCGTG
 28741 TATCCATATG ACACGGAAC CGTCTCTCA ACTGTGCTT TTCTTACTCC TCCCTTTGTA
 28801 TCCCCCAATG GGTTCAGAA GAGTCCCCCT GGGGTACTCT CTTTGGCGCT ATCCGAACCT
 28861 CTAGTTACTC CCAATGGCAT GCTTGGCTC AAAATGGGCA ACGGCCTCTC TCTGGACGAG
 28921 GCCCGCAACC TTACCTCCCA AAATGTAAAC ACTGTGAGCC CACCTCTCAA AAAAACCAAG
 28981 TCACAACTAA ACCTGGAAAT ATCTGACCCC CTCACAGTTA CCTCAGAGGC CCTAAGTGTG
 29041 GCTGCCGCCG CACCTCTAAT GGTGCGGCGC AACACACTCA CCATGCAATC ACAGGCGCGG
 29101 CTAACCGTGC ACGACTCCAA ACTTAGCATT GCCACCCAG GACCCCTCAC AGTGTGAGAA
 29161 GGAAGCTAG CCGTGCAAC ATCAGGCCCC CTCACCACCA CCGATAGCAG TACCCTTACT
 29221 ATCACTGCTC CACCCCTCT AACTACTGCC ACTGTAGCT TGGGCATTGA CTTGAAAGAG
 29281 CCACTTTATA CACAAAATGG AAAACTAGGA CTAAGTACG GGGCTCCTTT GCATGTAACA
 29341 GACGACCTAA ACACCTTGAC CGTAGCAACT GGTCCAGGTG TGACTATTAA TAATACTTCC
 29401 TTGCAACTA AAGTTACTGG AGCCTTGGGT TTTGATTAC AAGGCAATAT GCAACTTAAT
 29461 GTAGCAGGAG GACTAAGGAT TGATTCTCAA AACAGACGCC TTATACTTGA TGTAGTTAT
 29521 CCGTTTGATG CTCAAAACCA ACTAAATCTA AGACTAGGAC AGGGCCCTCT TTTTATAAAC
 29581 TCAGCCCACT ACTTGGATAT TAACTACAAC AAAGGCCCTTT ACTTGTATTAC AGCTTCAAAC
 29641 AATTCCAAAA AGCTTGAGGT TAACCTAAGC ACTGCCAAGG GGTGTATGTT TGACGCTACA
 29701 GCCATAGCCA TTAATGCAGG AGATGGGCTT GAATTTGGTT CACCTAATGC ACCAAACACA
 29761 AATCCCCCTCA AAACAAAAAT TGGCCATGSC CTAGRAATTG ATTCAAACAA GGCTATGGTT

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FIGURE 22
(SHEET 9)

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29821 CCTAAACTAG GAACTGGCCT TAGTTTGGAC AGCACAGGTG CCATTACAGT AGGAAACAAA
29881 AATAATGATA AGCTAACTTT GTGGACCA CAAGCTCCAT CTCCTAACTG TAGACTAAAT
29941 CGAGAGAAAG ATGCTAAACT CACTTTGGTC TTAACAAAAT GTGGCAGTCA AATACTTGCT
30001 ACAGTTTCAG TTTTGGCTGT TAAAGGCAGT TTGGCTCCAA TATCTGGAAC AGTTCAAGT
30061 GCTCATCTTA TTATAAGATT TGACGAAAT GGAGTGCTAC TAAACAATTC CTTCCTGGAC
30121 CCAGAAATATT GGAACCTTAG AAATGGAGAT CTTACTGAAG GCACAGCCTA TACAAACGCT
30181 GTTGGATTTA TGCCTAACCT ATCAGCTTAT CCAAAATCTC ACGGTAAAAC TGCCAAAAGT
30241 AACATTGTCA GTCAAGTTTA CTTAAAGGGA GACAAAATA AACCTGTAAC ACTAACCATT
30301 ACACATAACG GTACACAGGA AACAGGAGAC ACAACTCCAA GTGCATCTC TATGTCAATT
30361 TCATGGGACT GGTCTGGCCA CAACACATT AATGAATAT TTGCCACATC CTCTTACACT
30421 TTTTCATACA TTGCCAAGA ATAAAGAATC GTTTGTGTTA TGTTTCAACG TGTTTATTTT
30481 TCAATTGCAG AAAATTTCAA GTCAITTTTC ATTCAAGTAG ATAGCCCCAC CACCACATAG
30541 TCTATACAGA TCACCGTACC TTAATCAAAC TCACAGAAC CCAGTATTCA ACCTGCCACC
30601 TCCTCTCCAA CACACAGAGT ACACAGTCTT TTCTCCCGG CTGGCTTTAA AAAGCATCAT
30661 ATCATGGGTA ACAGACATAT TCTTAGGTGT TATATTCCAC ACGTTTCTCT GTCCAGCCAA
30721 ACGCTCATCA GTGATATTAA TAAACTCCCC GGGCAGCTCA CTTAAGTTCA TGTGCTGTG
30781 CAGCTGCTGA GCCACAGGCT GCTGTCCAAC TTGCGGTTGC TTAACGGGCG GCGAAGGAGA
30841 AGTCCACGCC TACATGGGGG TAGAGTCATA ATGCTGCATC AGGATAGGGG GGTGCTGCTG
30901 CAGCAGCGCG CGAATAAACT GCTGCCCGCG CCGCTCCGTC CTGACGGAAT ACAACATGGC
30961 AGTGGTCTCC TCACGCGATG TTGCGACCGC CCGCAGCATA AGGCGCCTTG TCCTCCGGGC
31021 ACAGCAGCGC ACCCTGATCT CACTTAAATC AGCACAGTAA CTGCAGCACA GCACCAATAT
31081 ATTGTTCAAA ATCCCAACAGT GCAAGCGCT GTATCCAAAG CTCTAGCGCG GCACCAACAG
31141 ACCCAGCGCG CCATCATACC ACAAGCGCAG GTAGATTAAG TGGCGACCCC TCATAAACAC
31201 GCTGACATA AACATTACCT CTTTGGGCTT GTTGAATTC ACCACCTCCC GGTACCATAT
31261 AACCTCTGTA TTAACATGG CGCCATCCAC CACCATCTTA ACCAGCTGG CCAAAACCTG
31321 CCGCCCGGCT ATACACTGCA GGGAACCGGG ACTGGAACAA TGACAGTGA GAGCCACGGA
31381 CTGATCAACA TGGATCATCA TGCTCGTCTT GATATCAATG TTGGCACAAC ACAGGCACAC
31441 GTGCTACAC TTCCCTAGGA TTACAGCTC CTCGCGGTT AGAACCATAT CCCAGGGAAC
31501 AACCCATTCG TGAATCAGCG TAAATCCAC ACTGCAGGGA AGACCTCGCA CGTAATCTAC
31561 GTTGTGCAAT GTCAAAAGTGT TACATTGCGG CAGCAGCGGA TGATCTCCA GTATGTTAGC
31621 GCGGTTCTCT GTCTCAAAAG GAGGTAGACG ATCCCTACTG TACGGAGTGC GCGGAGACAA
31681 CCGGATCGT GTTGTGCTGA GTGTCTGCC AAATGGAACG CCGGACGTAG TCATATTTC
31741 TGACGCAAAA CCAGGTGCGG GCGTGACAAA CAGATCTGCG TCTCGGTCT CGCCGCTTAG
31801 ATGCTCTGTT GTAGTAGTTG TAGTATATCC ACTCTCTCAA AGCATCCAGG CGCCCTCTGG
31861 CTTCCGGTTC TATGTAACT CTTTCATGCG CCGCTGCCCT GATAACATCC ACCACCGCAG
31921 AATAAGCCAC ACCCAGCCAA CCTACACATT CGTCTGCGA GTCCACACAG GGAGGAGCGG
31981 GAAGAGCTGG AAGAACCATG TTTTTTTTTT TATTCCAAA GATTATCCAA AACCTCAAAA
32041 TGAAGATCTA TTAAGTGAAC GCGCTCCCTT CCGGTGGCGT GGTCAAACTC TACAGCCAAA
32101 GAACAGATAA TGGCAITTTG AAGATGTTGC ACAATGGCTT CCAAAAGGCA AACCGCCTC
32161 ACGTCCAACT GGCAGTAAAG GCTAAACCTT TCAGGGTGA TCTCTCTAT AAACATTCCA
32221 GCACCTTCAA CCATGCCCAA ATAAATCTCA TCTGCCACC TTCTCAATAT ATCTCTAAGC
32281 AAATCCCGAA TATTAAGTCC GGCATTGTA AAATCTGCT CCAGAGCCCC CTCCACCTTC
32341 AGCCTCAAGC AGCGAATCAT GATTGCAAAA ATTCAGGTTT CTCACAGACC TGTATTAAGT
32401 TCAAAAGCGG AACATTAAAC AAAATACGCG GATCCGCTAG GTCCCTTCG GGGCCAGCT
32461 GAACATAATC GTGCAGTCT GCACGGACCA GCGCGGCCAC TTCCCGGCCA GGAACCTTGA
32521 CAAAGAACCC CACACTGATT ATGACACGCA TACTCGGAGC TATGCTAACC AGCGTAGCCC
32581 CGATGTAAGC TTGTGTGAT GGGCGGCGAT ATAAATGCA AGGTGCTGCT CAAAAATCA
32641 GGCAGAGCCT CGCGCAAAA AGAAGCACCA TCGTAGTCAT GCTCATGCAG ATAAAGGCG
32701 GTAAAGCTCC GAACACCCAC AGAAGAGAC ACCATTTTTT TCTCAACAT GTCTGCGGGT
32761 TCTGCATAA ACACAAAATA AAATAACAAA AAAACATTTA AACATTAGAA GCCTGTCTTA
32821 CAACAGGAAA AACRACCTT ATAGCATAA GACGGACTAC GGCATGCGG GCGTAGCCGT
32881 AAAAAAAGT GTACCGTGA TTAAGAGCA CCACCGACAG CTCTCGGTC ATGTCCGGAG
32941 TCATAATGTA AGACTCGTA AACACATCAG GTTGATTCTT CCGTCAGTGC TAAAAAGCGA
33001 CCGAATATAG CCGGGGGAAT ACATACCGCG AGGCGTAGAG ACAACATTAC AGCCCCATA
33061 GAGGTATATA CAAAATTAAT AGGAGAGAAA AACACATAAA CACCTGAAAA ACCCTCCTGC
33121 CTAGGCAAAA TAGCACCTC CCGCTCCAGA ACAACATACA GCGCTTCACA GCGGACGCT
33181 AACAGTCAGC CTTACAGTA AAAAGAAAA CCTATTAATA AACACCACT CGACACGGCA

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FIGURE 22
(SHEET 10)

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33241 CCAGCTCAAT CAGTCACAGT GTAAAAAGG GCCAAGTGCA GAGCGAGTAT ATATAGGACT
33301 AAAAAATGAC GTAAACGGTTA AAGTCCACAA AAAACACCCA GAAAACCGCA CGCGAACCTA
33361 CGCCCAGAAA CGAAAGCCAA AAAACCCACA ACTTCCTCAA ATCGTCACTT CCGTTTCCC
33421 ACGTTACGTA ACTTCCCAT TTAAGAAAAC TACAATTCCC AACACATACA AGTACTCCG
33481 CCCTAAAACC TAGTCAACC GCCCGTCC CACGCCCGC GCCACGTCAC AAATCCACC
33541 CCTCATTAT CATATTGGCT TCAATCCAA ATAAGGTATA TTATTGATGA TG

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FIGURE 22
(SHEET 11)

11

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LOCUS KD3 34341 bp DNA SYN 06-FEB-1999
DEFINITION KD3
ACCESSION KD3
KEYWORDS
SOURCE Unknown.
ORGANISM Unknown
Unclassified.
REFERENCE 1 (bases 1 to 34341)
AUTHORS Self
JOURNAL Unpublished.
FEATURES Location/Qualifiers
CDS
1..34341
/gene="KD3"
/product="KD3"
BASE COUNT 7951 a 9671 c 9464 g 7255 t
ORIGIN
1 CATCATCAAT AATATACCTT ATTTTGGATT GAAGCCAATA TGATAATGAG GGGGTGGAGT
61 TTGTGACGTG GCGCGGGCGG TGGGAACGGG GCGGTTGACG TAGTAGTGTG GCGGAAGTGT
121 GATGTTGCAA GTGTGGCGGA ACACATGTAA GCGACGGATG TGGCAAAAGT GACGTTTTTG
181 GTGTGCGCCG GTGTACACAG GAAGTGACAA TTTTCGCGCG GTTTTAAGCG GATGTTGTAG
241 TAAATTGCGG CGTAACCGAG TAAGATTGGG CCATTTTCGC GGGAAAACGT AATAAGAGGA
301 AGTGAAATCT GAATAATTTT GTGTACTCA TAGCGCGTAA TATTGTCTA GGGCCGCGGG
361 GACTTTGACC GTTTACGTGG AGACTCGCCC AGGTGTTTTT CTCAGGTGTT TTCCGCGTTC
421 CCGGTCAAAG TTGGCGTTTT ATTATTATAG TCAGCTGACG TGTAGTGTAT TTATAACCGG
481 TGAGTTCCTC AAGAGGCCAC TCTTGAGTGC CAGCGAGTAG AGTTTTCTCC TCCGAGCGCG
541 TCCGACACCG GGAAGTAAAA TGAGACATGA GGTACTGGCT GATAATCTTC CACCTCCTAG
601 CCATTTTGAA CCACCTACCC TTCACGAAGT GTATGATTTA GACGTGACGG CCCCAGGA
661 TCCCAACGAG GAGGCGGTTT CGCAGATTIT TCCCGACTCT GTAAATGTTG CGGTCCAGGA
721 AGGGATTGAC TTACTCACTT TTCGCGCGCG GCGCGGTTCT CCGGAGCGCG CTCACCTTTC
781 CCGGCGAGCC GAGCAGCGCG AGCAGAGAGC CTTGGGTCCG GTTTGCCACG AGGCTGGCTT
841 TCCACCCAGT GACGACGAGG ATGAAGAGGG TGAGGAGTTT GTGTTAGATT ATGTGGAGCA
901 CCGCGGCCAC GGTTCAGGTT CTTGTCTATA TCACCGGAGG AATACGGGGG ACCCAGATAT
961 TATGTGTTGG CTTTGTCTATA TGAGGACCTG TGCGATGTTT GTCTACAGTA AGTGAATAAT
1021 ATGGGCGAGT GGTGATAGAG TGGTGGGTTT GGTGTGGTAA TTTTTTTTTT AATTTTTTACA
1081 GTTTTGTGTT TTAAGAAATT TTGTATTGTT ATTTTTTTAA AAGGTCTCTG GTCTGAACCT
1141 GAGGCTGAGC CCGAGCCAGA ACCCGAGGCT GCAGAGCCTA CCGCGCGTCC TAAATGGCG
1201 CCTGCTATCC TGAGACGCCC GACATCACTT GTGCTAGAG AATGCAATAG TAGTACGGAT
1261 AGCTGTGACT CCGGTCCTTC TAACACACCT CCGTGAATAC ACCCGGTGTT CCGCTGTGTC
1321 CCCATTAAAC CAGTTGCCGT GAGAGTTGTT GGGCGTCGCC AGGCTGTGGA ATGTATCGAG
1381 GACTTGCTTA ACGAGCCTGG GCACCTTTTG GACTTGAGCT GTAAACGCCC CAGGCCATTA
1441 GGTGTAACCC TGTGATTGCG TGTGTGTTA ACGCCTTTGT TTGCTGAATG AGTTGATGTA
1501 AGTTTAATAA AGGTTGAGAT AATGTTTAA TGCATGCGCG TGTAAATGG GCGGGGCTT
1561 AAAGGTTATA TAATGCGCGG TGGGCTAATC TTGGTTACAT CTGACCTCAT GAGGCTTGG
1621 GAGTGTGTTG AAGATTTTTC TGCTGTGCGT AACTTGCTGG AACAGAGCTC TAACAGTACC
1681 TCTTGGTTTT GGAGGTTTTT GTGGGGCTCA TCCAGGCCAA AGTTAGTCTG CAGAATTAAG
1741 GAGGATTACA AGTGGGAATT TGAAGAGCTT TTGAATCCT GTGGTGAGCT GTTTGATTCT
1801 TTGAATCTGG GTCACCAAGC GCTTTTCCAA GAGAAGTCA TCAAGACTTT GGATTTTTCC
1861 ACACCGGGCG GCGCTGCGGC TGCTGTGCTT TTTTGTAGTT TTATAAGGA TAAATGGAGC
1921 GAAGAAACCC ATCTGAGCGG GGGGTACTCG CTGGATTTTC TGGCCATGCA TCTGTGAGA
1981 GCGGTTGTGA GACACAAGAA TCGCCTGCTA CTGTGTTCTT CCGTCCCGCC GCGGATAATA
2041 CCGACGAGAG AGCAGCAGCA GCAGCAGGAG GAAGCCAGCG GCGCGCGGCA GAGCAGAGC
2101 CCATGGAACC CGAGAGCCCG CCTGGACCTT CCGGAATGAA TGTGTACAG GTGGCTGAAC
2161 TGTATCCAGA ACTGAGACGC ATTTTGACAA TTACAGAGGA TGGGAGGGGG CTAAGGGGGG
2221 TAAAGAGGGA GCGGGGGGCT TGTGAGGCTA CAGAGGAGGC TAGGAATCTA GCTTTTAGCT
2281 TAATGACCA ACACCGTCTT GAGTGTATTA CTTTCAACA GATCAAGGAT AATTGCGCTA
2341 ATGAGCTTGA TCTGCTGGCG CAGAAGTATT CCATAGAGCA GCTGACCACT TACTGGCTGC
2401 AGCCAGGGGA TGATTTTGAG GAGGCTATTA GGGTATATGC AAAGTGGCA CTTAGGCCAG

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FIGURE 23
(SHEET 1)

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2461 ATTGCAAGTA CAAGATCAGC AAACCTGTAA ATATCAGGAA TTGTGTCTAC ATTTCTGGGA
2521 ACGGGGCCGA GGTGGAGATA GATACGGAGG ATAGGGTGGC CTTTAGATGT AGCATGATAA
2581 ATATGTGGCC GGGGGTGCTT GGCATGGAGG GGGTGGTTAT TATGAATGTA AGGTTTACTG
2641 GCCCAATTT TAGCGGTACG GTTTTCTGG CCAATACCAA CCTTATCCTA CACGGTGTA
2701 GCTTCTATGG GTTTAACAAT ACCTGTGTGG AAGCCTGGAC CGATGTAAAG GTTCGGGGCT
2761 GTGCTTTTGA CTGCTGTGG AAGGGGGTGG TGTGTGCGCC CAAAAGCAGG GCTTCAATTA
2821 AGAAATGCCT CTTTGAAGG TGTACCTGG GTATCTGTCT TGAGGGTAAC TCCAGGGTGC
2881 GCCACAATGT GGCCTCCGAC TGTGTTGCT TCAATGCTAGT GAAGAAGCGT GCTGTGATTA
2941 AGCATAACAT GGTATGTGGC AACTGCGAGG ACAGGGCCCTC TCAGATGCTG ACCTGTCTGG
3001 ACGGCAACTG TCACCTGCTG AAGACCATTG ACGTAGCCAG CCACTCTCCG AAGGCCTGGC
3061 CASTGTTTGA GCATAACATA CTGACCCGCT GTTCCTTGA TTTGGGTAAC AGGAGGGGGG
3121 TGTCTCTACC TTACCAATGC AATTTAGATC ACATAAGAT ATTGCTTAG CCCGAGAGCA
3181 TGTACAGTGA GACCTGAAC GGGGTGTTG ACATGACCAT GAAGATCTGG AAGGTGCTGA
3241 GGTACAGTGA GACCCGAC AGGTGCGAGC CCTGCGAGTG TGGCGGTAAA CATATTAGGA
3301 ACCAGCCTGT GATGCTGGAT GTGACCGAGG AGCTGAGGCC CGATCACTTG GTGCTGGCCT
3361 GCACCCGCGC TGAGTTTGGC TTAGCGATG AAGATACAGA TTGAGGTACT GAAATGTGTG
3421 GCGCTGGCTT AAGGGTGGGA AAGAATATAT AAGGTGGGG TCTTATGTAG TTTTGTATCT
3481 GTTTTGCAGC AGCCGCGCGC GCCATGAGCA CCAACTCGTT TGATGGAAGC ATTTGTAGCT
3541 CATATTGTAC AACCGCGATG CCCCCATGG CCGGGGTGCG TCAGAAATGT ATGGGCTCCA
3601 GCATTGTAGG TCGCCCGTGC CTGCCCCGAA ACTCTACTAC CTTGACCTAC GAGACCGTGT
3661 CTGGAACGCC GTTGGAGACT GCAGCCTCCG CCGCCGCTTC AGCCGCTGCA GCCACCGCCC
3721 GCGGGATTGT GACTGACTTT GCTTCTCTGA GCCCGCTTGC AAGCAGTGCA GCTTCCGTT
3781 CATCCGCGCG CGATGACAAG TTGACGGCTC TTTTGGCACA ATTGATTTCT TTGACCGGG
3841 AACTTAATGT CGTTTCTCAG CAGCTGTGG ATCTGCGCCA GCAGGTTTCT GCCCTGAAGG
3901 CTTCTCCTCC TCCCAATGCG GTTTAAACA TAAATAAAAA ACCAGACTCT GTTTGGATT
4021 GGTACCAAGCA AGTGTCTTGC TGTCTTTATT TAGGGGTTT CCGCGCGCGG TAGGCCGGG
4081 TCTGTATGTT CAGATACATG GGCATAAGCC CGTCTCTGGG GTGGAGGTAG CACCACTGCA
4141 GAGCTTCTATG CTGCGGGTGG GTGTGTAGA TGATCCAGTC GTAGCAGGAG GCGTGGCGT
4201 GGTGTCTAAA AATGTCTTTC AGTAGCAAGC TGATGCCAG GGGCAGGCC TTGGTGTAA
4261 TGTTTACAAA GCGGTTAAGC TGGGATGCGT GCATACGTGG GGATATGAGA TGCATCTTGG
4321 ACTGTATTTT TAGGTGGCT ATGTTCCAG CCAATCCCT CCGGGATTTC ATGTTGTGCA
4381 GAACACCAGC CACAGTGTAT CCGGTGCACT TGGGAAATTT GTCATGTAGC TTAGAAGGAA
4441 ATGCGTGGAA GAACTTGGAG ACGCCCTTGT GACCTCCAAG ATTTTCCATG CATTCGTCCA
4501 TAATGATGGC AATGGGCCCA CCGGCGCGCG CCGGGCGGAA GATATTTCTG GGTACTACTA
4561 CGTCATAGTT GTGTTCCAGG ATGAGATCGT CATAGCCAT TTTTACAAAG CCGGGCGGGA
4621 GGTGTGCCAG CTGCGGTATA ATGTTCCAT CCGGCCAGG GCGGTAGTTA CCCTCACAGA
4681 TTTGCAATTC CCACGCTTTC AGTTCCAGATG GGGGGATCAT GTCTACCTGC GGGGCGATGA
4741 AGAAACGGT TTCCGGGTA GGGGAGATCA GCTGGGAAGA AAGCAGGTTT CTGAGCAGCT
4801 GCGACTTACC GCAGCGGTG GGGCCGTAAA TCACACTAT TACCGGTTG AACTGTAGT
4861 TAAGAGAGCT GCAGCTGCCG TCATCCCTGA GCAGGGGGC CACTTCGTTA AGCATGTCCC
4921 TGACTCGCAT GTTTCCCTG ACCAAATCCG CCAGAAGCGG CTCGCGGCC AGCGATAGCA
4981 GTTCTTGCAA GGAAGCAAAG TTTTCAACG GTTTGAGACC GTCCGCGTA GGCATGCTTT
5041 TGAGCGTTTG ACCAAGCAGT TCCAGGCGGT CCCACAGCTC GGTCACTGCT TCTAGCGCAT
5101 CTGATCCAG CATATCTCCT CGTTTCCGG GTTGGGGCGG CTTTTCGCTGT ACGGCACTAG
5161 TCGGTGCTCG TCCAGACGGG CCGGCTCAT GTCTTCCAC GGGCGCAGG TCCTCTCAG
5221 CGTAGTCTGG GTCAAGGTGA AGGGGTGCGC TCCGGCTGC CCGGTCTTCG CCCTGCGCGT CCGCCAGGTA
5281 GAGGCTGGTC CTGCTGGTGC TGAAGCGCTG CCGGTCTTCG CCGTCCGCTG CCGCCAGCTT
5341 GCATTTGACC ATGTTGTCTAT AGTCCAGCCC CTCCGCGCGG TGCCCTTGG CCGCCAGCTT
5401 GCGCTTGAAG GAGGCGCCGC ACGAGGGGCA GTGCAGACTT TTGAGGGCGT AGAGCTTGGG
5461 GCGAGAGAA ACAGATTCCG GGGAGTAGGC ATCCGCGCGG CAGGCCCGCG AGACGCTCTG
5521 GCATTTCCAG AGCCAGGTGA GCTCTGGCCG TTCGGGGTCA AAAACAGGT TTCCCCATG
5581 CTTTGTATG CGTTTCTTAC CTCTGTTTC CATGAGCCG TGTTCCAGCT CCGTGAAGAA
5641 AAGGCTGTCC GTGTCCCGGT ATACAGACTT GAGAGGCCGT TCCTCGAGCG GTGTTCCGCG
5701 GTCTCTCTCG TATAGAACT CGGACCACTG TGAGACAAAG GCTCGGCTCC AGGCCAGCAC
5761 GAAGGAGGCT AAGTGGGAGG GGTAGCGGTC GTTGTCCACT AGGGGGTCCA CTCGCTCCAG
5821 GGTGTGAAGA CACATGTCCG CCTCTTCGCG ATCAAGGAAG GTGATTGGTT TGTAGGTGTA

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FIGURE 23
(SHEET 2)

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5881 GGCCACGTGA CCGGTGTTC CTGAAGGGG GCTATAAAG GGGTGGGG CGCGTTCGT
5941 CTCACCTCTCT TCCGATCGC TGCTCGGAG GCGCAGCTGT TGGGTGAGT ACTCCCTCTG
6001 AAAAGCGGC ATGACTTCTG CGCTAAGATT GTCACTTTCC AAAACGAGG AGGATTTGAT
6061 ATTCACCTGG CCGCGGTGA TGCCCTTGAG GGTGGCCGCA TCCATCTGGT CAGAAAAGAC
6121 AATCTTTTGT TTGTCAAGCT TGGTGGCAA CGACCGTAG AGGGCGTTGG ACAGCAACTT
6181 GGCATGAGG CGCAGGGTT GGTTTTGTG GCGATCGCG CGCTCCTGG CCGGATGTT
6241 TAGCTGCACG TATTCCGCG CAACGCAACC CCATCGGGA AAGACGGTGG TGCGCTCGTC
6301 GGCACCAAGG TGACGCGCC AACCGCGTT GTGAGGGGT ACAAGGTCAA CGCTGGTGGC
6361 TACCTCTCCG CGTAGGCGCT CGTTGTCCA GCAGAGGCGG CCGCCCTTGC GCGAGCAGAA
6421 TGGCGTAGG GGGTCTAGCT CGCTTCGTC CGGGGGTCT CGCTCCACGG TAAAGACCCC
6481 GGCAGCAGG CGCGCGTGA AGTAGTCTAT CTTCATCCT TGCAAGTCTA GCGCTGCTG
6541 CCAATGCGCG GCGCAAGCG CGCGCTCGTA TGGGTGAGT GGGGACCCC ATGCAATGGG
6601 GTGGGTGAGG GCGAGGCGT ACATGCCGA AATGTCTAA ACGTAGAGG GCTCTCTAG
6661 TATTCCAAAG TATGTAGGT AGCATCTTCC ACCGCGATG CTGCGCGCA CGTAATCTTA
6721 TAGTTCGTGC GAGGAGCGA GGAGGTCTGG ACCGAGGTG CTACGGCGG GCTCTCTG
6781 TCGGAAGAG ATCTGCCCTGA AGATGGCATG TGAGTTGAT GATATGGTTG GACGCTGAA
6841 GACGTTGAAG CTGGCGTCTG TGAGACCTAC CGCTCCACG ACAGAGGAGG CGTAGGAGTC
6901 GCGCAGCTTG TTGACAGCT CGGCGGTGAC CTGCACGCT AGGCGCAGT AGTCCAGGT
6961 TTCTCTGATG ATGTCTACT TATCTGTCC CTTTTTCCT CACAGCTCGC GGTGAGGAG
7021 AAACCTCTCG CGGTCTTCC AGTACTCTTG GATCGGAAAC CCGTGGCCT CGGAAAGGTA
7081 AGAGCTTAGC ATGTAGAACT GGTGACGCG CTGTAGGCG CAGCATCTCT TTTCTACGG
7141 TAGCGCGTAT GCTTCCGCG CCTTCCGAG CGAGGTGTGG GTGAGCGCA AGGTGCTCTC
7201 GACCATGACT TTGAGTACT GGTATTTGAA GTCACTTCC TCGCATCCG CCGCTCTCCA
7261 GAGCAAAAG TCCGTGCGCT TTTTGAACG CGGATTGAG AGGGCGAAGG TGACATCGTT
7321 GAAGATATC TTTCCGCGC GAGGCATAAA GTTGGGTGG ATGCGGAAGG GTCCCGGAC
7381 CTCGGAAGG TTGTTAATTA CCGCGCGCG GAGCACGAT TCGTCAAGC CGTTGATGTT
7441 GTGGCCACA ATGTAAAGTT CCAAGAAGCG CGGATGCCC TTGATGAAG GCAATTTTTT
7501 AAGTCTCTCG TAGGTGAGCT CTTCAGGGA GCTGAGCCG TGCTCTGAAA GGGCCAGTC
7561 TGCAAGATGA GGGTTGGAAG CGACAAATGA GCTCCACAG TCACGGGCA TTAGCATTTG
7621 CAGGTGGTCC CGAAAGGTCC TAAACTGGC ACCTATGGC ATTTTTCTG GGTGATGCA
7681 GTAGAAAGTA AGCGGTCTT GTTCCAGCG GTCCCATCCA AGGTTGCGG CTAGGTCTCG
7741 CGCGGAGTC ACTAGAGGCT CATCTCCGC GAACCTCATG ACCAGCATGA AGGCAAGAG
7801 CTGCTTCCCA AAGGCCCCA TCCAAGTATA GGTCTCTACA TCGTAGTGA CAAAGAGAG
7861 CTGGTGGCA GGTGCGAGC CGATCGGGA GAACCTGATC TCCCGCACG AATTGGAGGA
7921 GTGGCTATTG ATGTGGTGA AGTAGAAGTC CCGGAGCGG CGCAGCACT CGTGTGGCT
7981 TTTGTAAAA CGTGGCAGT ACTGGCAGG GTGACGGG TGTAATCTCT GCAAGAGGTT
8041 GACCTGACGA CCGCGCACAA GGAAGCAGAG TGGGAATTTG AGCCCTCGC CTGGCGGTT
8101 TGGCTGGTGG TCTTCTACT CGGCTGCTT TCGTTGACG TCTGGCTGCT CGAGGGGAGT
8161 TACGGTGGAT CGGACCACTA CGCGCGCGA GCGCAAGTC CAGATGTCG CCGCGCGG
8221 TCGGAGCTTG ATGACAACAT CGCGCAGAT GAGAGCTGCC ATGGTCTGGA GCTCCCGCG
8281 CGTCAGGTC GCGCGAGCT CCGCAGGTT TACCTGCTAT AGACGGGTCA GCGCGCGG
8341 TAGATCCAGG TGATACCTAA TTTCCAGGG CTGGTGGTG CCGCGCTGA TGGCTGCAA
8401 GAGGCCGAT CCGCGCGG CGACTACGT ACCCGCGG GGGCGGTGG CCGCGGGGT
8461 GTCTTGGAT GATGATCTA AAAGCGTGA CGCGGCGAG CCGCGGAGG TAGGGGGG
8521 TCGGACCCG CCGGAGAGG GGGCAGGGC AGTCCGCG CCGCGCGG CAGGAGCTGG
8581 TGCTGCGCG GTAGTTGCT GCGCAACCG AGACGCGG GGTGATCTC CTGAATCTGG
8641 CGCCTCTGG TGAAGACGAC GGGCCCGTG AGCTTGAGC TGAAGAGAG TTGACAGAA
8701 TCAATTTGG TGTGTTGAC GCGCGCTGG CGCAAAATCT CCGTCACTG TCTGAGTTG
8761 TCTTGATAGG CGATCTCGG CATGAATGC TCGATCTCT CCGCTGAGG ATCTCCCGT
8821 CCGCTCGCT CCAAGGTGG GCGAGGTCG TTGGAATGC GGGCCATGAG CTGCGAGAAG
8881 CGCTTAGGCG CTCTCTGTT CCAGACGCG CTGTAGACCA CGCCCTTTC GGCATCGCG
8941 GCGCGCATGA CCACCTGCG GAGATTGAG TCCAGTGCC GGGCGAAGAC GCGTAGTTT
9001 GCGAGCGCT GAAAGAGGT GTTGAAGGT GTGCGGTGT GTTCTGCTC GAAGAGTAC
9061 ATACCCAGC GTGCAAGT GGAATCGTT ATATCCCCA AGGCTTCAAG GCGCTCCATG
9121 GCCTCTGTA AGTCCACGG GAAGTTGAAA AACTGGAGT TGCGCGCGA CAGGTTAAC
9181 TCCTCTCCA GAAGACGAT GAGCTCGCG ACAGTGTCG GCACCTCGG CTCAGAGGCT
9241 ACAGGGGCT CTCTCTCTC TTCAATCTC TCTTCCATA GGGCTTCCC TTCTCTCTC

9301 TCTGCGCGCG GTGGGGGAGG GGGGACACGG CGGCGACGAC GGGCGACCGG GAGGCGGTCC
9361 ACAAAGCGCT CGATCATCTC CCCGCGGCGA CGGCGCATGG TCTCGGTGAC GGGCGGGCCG
9421 TTCTCGCGCG GCGCGAGTTG GAAGACGCGG CCCGTATGT CCCGGTTATG GGTGCGCGCG
9481 GGGCTGCCAT GCGGCGAGGA TACGGCGCTA ACATGCATC TCAACAATTG TTGTGTAGGT
9541 ACTCCGCGCG CGAGGGACCT GAGCGAGTCC GCATCGACCG GATCGGAAAA CCTCTGAGA
9601 AAGCGCTCTA ACCAGTCACA GTGCAAGGT AGGCTGAGCA CGGTGGCGGG CGGCGAGCGG
9661 CGGCGGTCCG GGTGTTTCT GCGGAGGTG CTGCTGATGA TGTAAATAA GTAGGCGGTG
9721 TTGAGACGGG GGATGCTCGA CAGAAGCACC ATGTCTTGG GTCCGGCTG CTGAATGCGC
9781 AGGCGGTCCG CCATGCCCA GGCTTCGTTT TGACATCGGC GCAGTCTTT GTAGTAGTCT
9841 TGCAATGAGCC TTTCTACCG CACTTCTTCT TCTCCTTCT CTGTCTTCT ATCTCTTGCA
9901 TCTATCGCTG CGGCGGCGGC GGAGTTTGGC CGTAGGTGGC GCCTCTTCT TCCCATGCGT
9961 GTGACCCCGA AGCCCTCAT CGGCTGAAGC AGGCTAGGT CGGCGACAAC CGCTCGGCT
10021 AATATGCGCT GCTGCACCTG CGTGAGGTA GACTGGAAGT CATCCATGTC CACAAAGCGG
10081 TGGTATGCGC CCCTGTTGAT GGTGTAAGTG CAGTTGGCCA TAACGGACCA GTTAAACGGT
10141 TGGTGACCGC GCTGCGAGAG CTCGGTGTAC CTGAGACGCG AGTAAGCCCT CGAGTCAAT
10201 ACGTAGTCGT TGCAAGTCCG CACCAGGTAC TGGTATCCCA CCAAAAAGTG CGGCGCGCGC
10261 TGGCGGTAGA GGGGCCAGCG TAGGTTGGCC GGGGCTCCGG GGGCGAGATC TTCCACATA
10321 AGGCGATGAT ATCCGTAGAT GTACCTGAGC ATCCAGGTGA TGCCGGCGGC GTTGGTGGAG
10381 GCGCGCGGAA AGTCGCGGAC GCGGTTCCAG ATGTTGCGCA GCGGCAAAAA GTGCTCCATG
10441 GTCCGGAACG CTCGGCCGGT TCTGGTGGTC TGGTGGATAA ATTGCAAGG GTATCATGGC
10501 GAGCTCTGAA GCGGCGACTC TTCTGTGGT CAGTTGGATAA ATTGCAAGG GTATCATGGC
10561 GAGCGACCGG GGTTCGAGCC CGGTATCCGG CGGTCCGCGG TGTATCCATG GGTATCCGCC
10621 CGCGTGTGCA ACCCAGGTGT GCGACGTCAG ACACCGGGGG AGTGTCTCTT TTGCTTCTCT
10681 TCCAGGCGCG GCGGCTGCTG CGCTAGCTTT TTTGGCCACT GGGCGGCGCG AGCGTAAGCG
10741 GTTAGGCTGG AAGCGGAAAG CATTAACTGG CTCGCTCCCT GTAGCCGGAG GGTATTTTTC
10801 CAAGGGTTGA TCCGCGGAGC CCCGCTTCC AGTCTCGGAC CGGCGGACT CGGCGGAAAG
10861 GGGGTTTTCG TCCCGTCTAT GCAAGACCCC GCTTGCAGAT TCCTCCGAA ACAGGGACGA
10921 GCCCTTTTTT TGCTTTTCCC AGATGCATCC GGTGCTGCGG CAGATGCGCC CCCCTCTCA
10981 GCAGCGGCAA GAGCAAGAGC AGCGGCGAGC ATGCAAGGCA CCCTCCCTCT CTCCTAGCGC
11041 GTACGAGAGG GCGACATCCG CGGTTGACCG GGCAGCAGAT GGTGATTACG AACCCCGCGC
11101 GCGCGCGGCC GCGCACTACC TGGACTTGGG GAGGCGCGAG GGCCTGCGCG GGTAGGAGC
11161 GCCCTCTCTT GAGCGGTACC CAAGGGTGCA GCTGAAGCGT GATACGCGTG AGGCGTACGT
11221 GCGCGCGCAG AACCTGTTTC GCGACCGCGA GGGAGAGGAG CCGAGGAGA TGCGGGATCG
11281 AAAGTTCCAC GAGGGCGCG AGCTGCGGCA TGGCTGAAAT CGCGAGCGGT TGCTGCGCGA
11341 GGAGGACTTT GAGCCCGAGC CGCGAACCGG GATTAGTCCC CGCGCGCGAC ACCTGCGCGC
11401 CGCCGACCTG GTAACCGCAT ACGAGCAGAC GGTGAACCGG GGTGATTACG AACCCCGCGC
11461 CTTTAAACAC CACGTGCGTA CGCTTGTGGC GCGCGAGGAG GTGGCTATAG GACTGATGCA
11521 TCTGTGGGAC TTTGTAAGCG CGCTGAGGCA AAACCCAAAT AGCAAGCGCG TCATGGCGCA
11581 GCTGTCTCTT ATAGTGAGC ACAGCAGGGA CAACGAGGCA TTCAGGATG CGCTGCTAAA
11641 CATAGTAGAG CCCAGGGCG GCTGGCTGCT CGATTGATA AACATCTGCG AGAGCATAGT
11701 GGTGACAGAG CGCAGCTTGA GCTGCGTGA CAAGGTGGCC GCCATCAACT ATTCCATGCT
11761 TAGCCTGGGC AAGTTTTACG CCCGCAAGAT ATACCATACC CTTAGCTTC CCATAGACAA
11821 GGAGGTAAG ATCGAGGGGT TCTACATGCG CATGGGCGTG AAGGTGCTTA CCTTGAGCGA
11881 CGACCTGGGC GTTTATCGCA ACGAGCGCAT CCACAAGGCC GTGAGCGTGA GCGGCGCGCG
11941 CGAGCTCAGC GACCGGAGC TGATGCACAG CCTGCAAGG GCCTGCGCTG GCACGGGCGG
12001 CGGCGATAGA GAGGCGAGT CTTACTTTGA CGCGGGCGCT GACCTGCGCT GGGCCCCAAG
12061 CGGACGCGCC CTGGAGGCG CTGGGGCGCG ACCTGGGCTG CGGCTGGCAC CCGCGCGCGC
12121 TGGCAACGTC GCGGCGGTGG AGGAATATGA CAGGAGCGAT GAGTACGAGC CAGAGGACGG
12181 CGAGTACTAA CCGGTGATGT TTCTGATCAG ATGATGCAAG ACGCAACGGA CCCGCGGTG
12241 CGGCGCGCGC TGCAGAGCCA GCGTCCGCG CTTAACTCCA CGGACGACTG GCGCCAGGTC
12301 ATGAGCCGCA TCATGTCGCT GACTGCGCGC AATCTGAGC CGTTCCGCGA GCGCCGCGC
12361 GCGAACCGCG TCTCCGCAAT TCTGGAAGCG GTGGTCCCGG CGCGCGCAAA CCGCACGCGC
12421 GAGAAGGTGC TGGCGATCGT AAACGCGCTG GCGGAAACA GGGCCATCCG GCGCGAGGAG
12481 GCGCGCTGCG TCTACGAGC GCTGCTTCAG CCGGTGGCTC GTTACCAACG CCGCAACGCG
12541 CAGACCAACC TGGACCGGCT GGTGGGGGAT GTGCGCGAGG CGGTGGCGCA GCGTGAAGCG
12601 GCGCAGCAGC AGGCGCAACT GGGCTCCATG GTTGCATTA ACCTCTCTCT GAGTACACAG
12661 CCGCCCAACG TCGCGCGGGG ACAGGAGGAC TACACCAACT TTGTAGAGCG ACTGCGGCTA

12721 ATGGTGACTG AGACACCGCA AAGTGAGGTG TACCAGTCTG GGCAGACTA TTTTTCACG
 12781 ACCAGTAGAC AAGGCTTGCA GACCGTAAAC CTGAGCCAGG CTTTCAAAAA CTTGCAGGGG
 12841 CTGTGGGGGG TGCGGGCTCC CACAGGCGAC CGCGCGACCG TGTTAGCTT GCTGACGCCC
 12901 AACTGGCGCC TGTTGCTGCT GCTAATAGCG CCGTTACGG ACAGTGGCAG CGTGTCCCGG
 12961 GACACATACC TAGGTCACTT GCTGACACTG TACCGCGAGG CCATAGGTCA GGCACATGTG
 13021 GACGAGCATA CTTTCCAGGA GATTACAAGT GTCAGCGCGG CGCTGGGGCA GGAGGACACG
 13081 GGCAGCCTGG AGGCAACCTT AAACCTACTG CTGACCAACC GGCAGCAGAA GATCCCTCTG
 13141 TTGCACAGTT TAAACAGCGA GGAGGAGCGC ATTTTCCGCT ACCTGCAGCA GAGCCTGAGC
 13201 CTTAACCTGA TGCGCGACGG GGTAAACGCC AGCGTGGCGC TGGACATGAC CGCGCGCAAC
 13261 ATGGAAACCG GCATGTATGC CTCAAACCGG CCGTTTATCA ACCGCTTAAT GGACTACTTG
 13321 CATCGCGCGG CCGCCGTGAA CCGCGAGTAT TTCACCAATG CCATCTTGAA CCGCACTGG
 13381 CTACCGCTCC CTGTTTCTA CACCGGGGGA TTCAGGTGTC CCGAGGGTAA CGATGGATTG
 13441 ACTCGGAGCG ACATAGACGA CAGCGTGTTC TCCCGCAAC CGCAGACCTT GCTAGAGTTG
 13501 CAACAGCGCG AGCAGGCGA GCGCGGCTG CGAAAGGAAA GCTTCCGCG GCGAAGCAGC
 13561 TTGTCGATC TAGGCGCTGC GCGCCCGCGG TCAGATGCTA GTAGCCCAT TCCAGCTTG
 13621 ATAGGGTCTC TTACAGCAC TCGCACACC CCGCCGCGCC TGCTGGGCGA GGAGGAGTAC
 13681 CTAACAACCT CGCTGCTGCA GCGCGAGCGC GAAAGAAACC TGCTCCGCG ATTTCCCAAC
 13741 AACGGGATAG AGAGCCTAGT GACCAAGATG AGTAGATGGA AGACGTACGC GCAGGAGCAC
 13801 AGGACGCTGC CAGGCGCGCG CCGCGCCACC CGTCTGCTAA GGCACGACCG TCAGCGGGT
 13861 CTGGTGTGGG AGGACGATGA CTGGCGAGAC GACAGCAGCG TCCTGGATT TGGAGGGAGT
 13921 GGCACCCGCT TTGCGCACTT TCGCCCGAGG CTGGGGAGAA TGTTTTAAAA AAAAAAAGC
 13981 ATGATGCAAA ATAAAAACT CACCAAGGCC ATGGCACCGA CGGTTGGTTT TCTGTATTG
 14041 CCCTTAGTAT CGGCGCGCGG GCGATGTATG AGGAAGGTCC TCCTCCCTCC TACGAGAGTG
 14101 TGTTGAGCGC GCGCGCAGTG GCGCGCGCGC TGGGTTCTCC CTTGATGCT CCCCTGGACC
 14161 CGCGGTTTGT GCGCTCGCGG TACCTGCGGC CTACCGGGGG GAGAAACAGC ATCCGTTACT
 14221 CTGAGTTGGC ACCCTATTTC GACACCAACC GTGTGTACCT GGTGGACAC AAGTCAACGG
 14281 ATGTGGCATC CTTGAACCTAC CAGAACGACC ACAGCAACTT TCTGACCAAG GTCAATCAAA
 14341 ACATGACTA CAGCCCGGGG GAGGCAAGCA CACAGACCAT CAATCTTGAC GACCGGTGCG
 14401 ACTGGGCGCG CGACCTGAAA ACCATCTGCG ATACCAACAT GCCAATGTG AACGAGTTCA
 14461 TGTTTACCAA TAAGTTTAA GCGCGGGTGA TGTTGTCGCG CTTGCTACT AAGGACATC
 14521 AGTGTGAGCT GAAATACGAG TGGGTGGAGT TCAGCTGCTC CGAGGGCAAC TACTCCGAGA
 14581 CCATGACCAT AGACCTTATG AACAAACGGA TCCTGGAGCA CTACTTGAAA GTGGGACAGC
 14641 AGAACGGGGT TCTGGAAAGC GACATCGGGG TAAAGTTTGA CACCGCAAC TTCAGACTGG
 14701 GGTGAGGCC CGTCACTGGT CTTGTCTATG CTGGGGTATA TACAAACGAA GCCTTCCATC
 14761 CAGACATCAT TTTGCTGCCA GATGCGGGG TGGACTTAC CCACAGCGCG CTGAGCAACT
 14821 TGTTGGGCAT CCGCAAGCGG CAACCTTCC AGGAGGGCTT TAGGATCACC TACGATGATC
 14881 TGGAGGGTGG TAACATTCCC GCACTGTTGG ATGTGGACGC CTACCAAGCG AGCTTGAAAG
 14941 ATGACACCGA ACAGGGCGGG GGTGGCGCAG GCGGCAGCAA CAGCACTGGC AGCGGCGCG
 15001 AAGAGAACTC CAACGCGGCA GCGCGGCAA TGCAGCCGGT GGAGGACATG AACGATCATG
 15061 CCATTTCGCG CGACACCTTT GCCACACGGG CTGAGGAGAA CGCGCTGAG GCCGAAGCAG
 15121 CGGCGGAAGC TGCGCGCCCC GCTGCGCAAC CCGAGGTGGA GAAGCCTCAG AAGAAACCGG
 15181 TGATCAAAAC CCTGACAGAG GACAGCAAGA AACGCACTTA CAACCTAATA AGCAATGACA
 15241 GCACCTTCAAC CCACTACCGC AGCTGTGACC TTGCATACAA CTACGGCGAC CCTCAGACCG
 15301 GAATCCGCTC ATGAGACCTG CTTTGCACTC CTGACGTAAC CTGCGGCTCG GAGCAGGTCT
 15361 ACTGGTCGTT GCCAGACATG ATGCAAGACC CCGTGAACCT CCGCTCCAGC CGCCAGATCA
 15421 GCAACTTTCC GGTGGTGGGC GCGAGCTGT TCGCGTGCA CTCCAAAGAGC TTCTACAACG
 15481 ACCAGGCGGT CTACTCCCAA CTCTATCCGC AGTTTACCTC TCTGAGCCAC GTGTTCAATC
 15541 GCTTTCCCGA GAACCAAGAT TTGGCGCGCC CGCCAGCCCC CACCATCACC ACCGTGAGTG
 15601 AAAACGTTCC TGCTCTCACA GATCACGGGA CGCTACCGCT GCGCAACAGC ATCGGAGGAG
 15661 TCCAGCGAGT GACCATTACT GACGCCAGAC GCGCACCTG CCGCTACGTT TACAAGGCCG
 15721 TGGGATAGT CTGCGCGCGG GTCTATCGA GCGGCACCTT TTGAGCAAGC ATGTCCATCC
 15781 TTATATGCCC CAGCAATAAC ACAGGCTGGG GCGTGGCTT CCGAAGCAAG ATGTTTGGG
 15841 GGGCCAAGAA GCGCTCCGAC CAACACCCAG TCGCGGTGCG CCGGCACTAC CGCGCGCCCT
 15901 GGGCGCGGCA CAACCGCGGC CGCACTGGGC GCACACCGGT CGATGACGCC ATCGACGCGG
 15961 TGTTGGAGGA GCGCGCAAC TACACGCCCA CGCGGCCACC AGTGTCCACA GTGGACGCGG
 16021 CCATTCAAGC CGTGGTGGC GAGGCCCGG GCTATGCTAA AATGAGAGA CGCGGAGGCG
 16081 CGGTAGCACG TCGCCACCGG CCGCGACCGG GCACTGCGCG CCAACGCGCG GCGCGCGCCC

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FIGURE 23
(SHEET 5)

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16141 TGCTTAACCG CGCACGTCCG ACCGGCCGAC GGGCGGCCAT GCGGGCCGCT CGAAGGCTCG
16201 CCGCGGGTAT TGTCACCTGTG CCCCCCAGGT CCAGGCGGACG AGCGGCGGCC GCAGCAGCCG
16261 CGGCATTAG TGCTATGACT CAGGCTCGCA GGGGCAACGT GTATTGGGTG CCGACTCTGG
16321 TTAGCGGCTT GCGCGTGCCC GTGCGCACCC GCGCCCGCG CAACTAGATT GCAAGAAAAA
16381 ACTACTTAGA CTGCTACTGT TGATGTATC CAGCGCGCGG GCGCGCGAAC GAACTATGT
16441 CCAAGCGCAA AATCAAAGAA GAGATGCTCC AGGTCAATCG GCGCGAGATC TATGGCCCCC
16501 CGAAGAAGSA AGAGCAAGAT TACAAGCCCC GAAAGCTAAA GCGGCTCAAA AAGAAAAAGA
16561 AAGATGATGA TGATGAACCT GAGCAGGAGG TGGAACTGCT GCACGCTACC GCGCCAGGC
16621 GACGGGTACA GTGGAAAGGT CGACGCTTAA AACGTGTATT GCGACCCGCG ACCACCTAG
16681 TCTTTACGCC CGGTGAGCGC TCCACCCGCA CCTACAAGCG CGTGTATGAT GAGGTGTACG
16741 GCGCAGAGGA CCGTCTTGAG CAGGCCAACG AGCGCTCGG GGAGTTTGCC TACGAAAGC
16801 GGCATAAGGA CATGCTGGCG TTGCGCTGG ACAGGGGCAA CCCAACACCT AGCCTAAAGC
16861 CCGTAACTACT CGACGAGGTG CTGCCCGCGC TTGCACCGTC CGAAGAAAAA GCGGCTTAA
16921 AGCGCGACTC TGCTGACTTG GCACCCACCG TGCAGCTGAT GGTACCCAA GCGCAGCGAC
16981 TGGAAAGTGT CTTGAAAAA ATGACCGTGG AACCTGGGCT GAGGCCGAG GTCCCGTGC
17041 GCGCAATCAA CGAGGTGGCG CCGGACTGG GCGTGACAGC CGTGGACGTT CAGATACCCA
17101 CTACCAAGTAG CACCAATATT GCCACCGCCA CAGAGGGCAT GGAGACAACA ACGTCCCGCG
17161 TTGCTCAGC GGTGGCGGAT GCGCGGTGC AGCGGTGCG TCGCGCCGCG TCCAAGACCT
17221 CTACGAGGT GCAACGGAC CCGTGTGATG TTGCGGTTT AGCCCCCGG GCGCGCGCG
17281 GTTCGAGGAA GTACGGCGCC GCCAGCGCGC TACTGCGCGA ATATGCCCTA CATCTTCCA
17341 TTGCGCTTAC CCGCGCTAT CCGTGTGACA CCTACCGCCC CAGAAGACGA GCACTACCC
17401 GAGCGCGAAC CACCACTGGA ACCCGCGGCC GCGCTCGGCC TCGCCAGCC CGTGGTGTG
17461 CGATTTCGCT GCGCAGGCTG GCTCGCGAAG GAGGCAGGAC CCTGGTGTG CCAACAGCGC
17521 GCTACCAACC CAGCATCGTT TAAAGCCGCG TCTTTGTGTT TCTTGACGAT ATGGCCCTCA
17581 CCGCGCGCT CCGTTTCCCG GTGCGGGAT TCCGAGGAAG AATGCACCGT AGGAGGGGCA
17641 TGGCGCGCCA CGGCTGACG GCGCGCATGC GTGCTGGCG CCACCGCGCG GCGCGCGCT
17701 CGCAGCGCTG CATGCGCGCG GGTATCTGTC CCTCTCTTAT TCCACTGATC GCGCGCGCGA
17761 TTGCGCGCTT GCGCGGAAT GCATCCGTGG CCTTGACGCG GCAGAGACAC TGATTAAAAA
17821 CAAGTTGCTAT GTGAAAAAT CAAAAATAAA AGTCTGACT CTCAAGCTCG CTTGCTCTG
17881 TAATATTATT GTAGAAATGA AGACATCAAC TTTGCGTCTC TGGCCCCGCG ACACGCTCG
17941 CGCCCGTTCA TGGGAAACTG GCAAGATATC GGCACAGCA ATATGAGCG TGGCGCTTC
18001 AGCTGGGCT CCGTGTGGAG CGGCATTAAA AATTTCGTTT CCACCGTTAA GAACTATGCG
18061 AGCAAGGCTT GGAACAGCAG CACAGGCCAG ATGCTGAGGG ATAAGTTGAA AGAGCAAAAT
18121 TTCCAACAAA AGGTGGTAGA TGGCCTGGCC TCTGGCATT GCGGGGTGTT GGACCTGGCC
18181 AACCGGCGAG TGCAAAATAA GATTAAACAGT AAGCTTGATC CCGCGCTCC CGTAGAGGAG
18241 CCTCACCGCG CCGTGGAGAC AGTGTCTCCA GAGGGGCGTG GCUAAAAGCG TCGCGCGCCC
18301 GACAGGGAAG AAACCTCTGT GACGCCAATA GACGAGCCTC CCTCGTACGA GGAGGCACTA
18361 AAGCAAGGCC TGCCACCAC CCGTCCCATC GCGCCCATGG CTACCGAGT GCTGGGCCAG
18421 CACACACCGG TAAACGCTGA CCGTCCCTCC CCGCGCGACA CCCAGCAGAA ACCTGTGCTG
18481 CCGAGGCCGA CCGCGCTGTG TGTAAACCGT CCTAGCGCG CGTCCCTGCG CCGCGCGCCC
18541 AGCGGTCCGC GATCGTTGCG GCCCGTAGCC AGTGGCAACT GGCAGAGCAC ACTGAACAGC
18601 ATCGTGGGTG TGGGGGTGCA ATCCCTGAAG CCGCGAAGAT GCTTCTGAAT AGCTAACGTG
18661 TCGTATGTGT GTCATGTATG CGTCCATGTC GCGCGCAGAG GAGCTGCTGA GCGCGCGCGC
18721 GCGCGCTTTC CAAGATGCTT ACCCCTTCCA TGATGCCGCA GTGCTTCTT ATGCACATCT
18781 CCGGCCAGGA CGCCTCGAG TACCTGAGCC CCGGCTGTGT GCAGTTTTC CCGGCCACG
18841 AGACGTACTT CAGCCTGAAT AACAGTTTA GAAACCCAC GGTGGCGCTG ACGCACGAGC
18901 TGACACAGA CCGTCCAG CGTTTGACCG TCGGTTTCTT CCGTGTGAC CCGTGGGATA
18961 CTGCGTACTC GTACAAGCG CGGTTCAACC TAGCTGTGGG TGATAACCGT GTGCTGGACA
19021 TGGCTTCCAC GTACTTTGAC ATCCGCGCGC TGCTGACAG GGGCCCTACT TTAAAGCCCT
19081 ACTCTGGCAC TGCTACAAC GCGCTGGCTC CCAAGGGTGC CCCAATCCT TCGAATGGG
19141 ATGAAGCTGC TACTGCTCTT GAAATAAAC CTACGCTATT TGGCGAGCG CTTATTCTG
19201 AAGTAGACGA GCAAGCTGAG CAGCAAAAA CTACGCTATT TGGCGAGCG CTTATTCTG
19261 GTATAAATAT TACAAGGAG GGTATTCAA TAGGTGTGCA AGGTCAAACA CTTAAATATG
19321 CCGATAAACC ATTTCAACCT GAACCTCAA TAGGAGATC TCAGTGGTAC GAACTGAAA
19381 TTAATCATGC AGCTGGGAGA GTCTTAAAA AGACTACCCC AATGAAACCA TTTTACGTT
19441 CATATGCAAA ACCCACAAT GAAATGGAG GGAAGGCAT TCTTGTAAAG CAACAAAATG
19501 GAAAGCTAGA AAGTCAAGTG GAAATGCAAT TTTTCTCAAC TACTGAGGCG ACCGACGCA

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FIGURE 23
(SHEET 6)

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19561 ATGGTGATAA CTTGACTCCT AAAGTGGTAT TGTACAGTGA AGATGTAGAT ATAGAAACCC
19621 CAGACACTCA TATTTCCTTAC ATGCCCACTA TTAAGGAAGG TAACACACGA GAACATAATGG
19681 GCCACCAATC TATGCCCAAC AGGCCTAATT ACATTGCTTT TAGGGACAAT TTTATTGGTC
19741 TAATGTATTA CAACAGCACG GGTAAATATGG GTGTTCTGGC GGGCCAAGCA TCGCAGTTGA
19801 ATGCTGTTGT AGATTGCAA GACAGAAACA CAGAGCTTTC ATACCAGCTT TTGCTTGATT
19861 CCATTGGTGA TAGAACCAGG TACTTTTCTA TGTGGAATCA GGCTGTTGAC AGCTATGATC
19921 CAGATGTTAG AATTATTGAA AATCATGGAA CTGAAGATGA ACTTCCAAT TACTGCTTTC
19981 CACTGGGAGG TGTGATTAA ACAGAGACTC TTACCAAGGT AAAACCTAAA ACAGGTCAGG
20041 AAAATGGATG GGAAGAAAGAT GCTACAGAAAT TTTCAGATAA AAATGAATA AGAGTTGAA
20101 ATAAATTTGC CATGGAATC AATCTAAATG CCAACCTGTG GAGAAATTTT CTGTACTCCA
20161 ACATAGCGCT GTATTGCCC GACAAGCTAA AGTACAGTCC TTCCAACGTA AAAATTTCTG
20221 ATAAACCCAA CACCTACGAC TACATGAACA AGCGAGTGGT GGCTCCCGGG TTATGGGACT
20281 GCTACATTAA CCTTGGAGCA CGCTGGTCCC TTGACTATAT GGACAACGTC AACCCATTTA
20341 ACCACCAACG CAATGCTGGC CTGCGCTACC GCTCAATGTT GCTGGCAAT GGTGCTATG
20401 TGCCCTTCCA CATCCAGGTG CCTCAGAAGT TCTTTGCCAT TAAAAACCTC CTCTCCTGC
20461 CGGGCTCATA CACCTACGAG TGGAACTTCA GGAAGGATGT TAACATGGTT CTGACAGCT
20521 CCCTAGGAAA TGACCTAAGG GTTGACGGAG CAGCATTAA GTTTGATAGC ATTTGCCCTT
20581 ACGCCACCTT CTTCGCCATG GCCACAACA CCGCTCCAC GCTTGAGGCC ATGCTTAGAA
20641 ACGACCCAA CGCTACCAAC GTGCCCATAT CCATCCCTC CCGCAACTGG CGGGCTTCC
20701 TACCCGCCAA CGCTACCAAC GTGCCCATAT CCATCCCTC CCGCAACTGG CGGGCTTCC
20761 GCGGCTGGGC CTTCACGCGC CTTAAGACTA AGGAAACCCC ATCACTGGGG TCGGGCTAAG
20821 ACCCTTATTA CACCTACTCT GGCTCTATAC CCTACTAGA TGGAACTTTT TACCTCAACC
20881 ACACCTTTAA GAAGGTGGCC ATTACCTTGG ACTCTTCTGT CAGCTGGCCT GGCATGACC
20941 GCTGCTTAC CCCCACGAG TTTGAATTA AGCGCTCAGT TGACGGGGAG GGTTACAACG
21001 TTGCCCACTG TAACATGACC AAAGACTGGT TCCTGTGACA AATGCTAGCT AACTACAACA
21061 TTGGCTACCA GGGCTTCTAT ATCCAGAGA GCTACAAGGA CCGCATGTAC TCCTTCTTTA
21121 GAACTTTCCA GCCCATGAGC CGTCAGGTGG TGGATGATAC TAAATACAAG GACTACCAAC
21181 AGGTGGGCAT CCTACACCAA CACAACAAC CTGGATTGTT TGGCTACCTT GCCCCACCA
21241 TGCGCGAAGG ACAGGCTTAC CCGCTATACC TCCCTATACC GCTTATAGG CCGCCACCTT
21301 TTGACAGCAT TACCCAGAAA AAGTTTCTTT GCGATCGCAC CCTTTGGCGC ATCCCATCTT
21361 CCGATAACTT TATGTCATG GCGCAGTCA CAGACCTGGG CCAAAACCTT CTCTACGCCA
21421 ACTCCGCCCA CGCGCTAGAC ATGACTTTTG AGGTGGATCC CATGGACGAG CCCACCTTTC
21481 TTTATGTTTT GTTTGAAGTC TTGACGTGG TCGGTGTGCA CCGCCCGCAC CGCGCGCTCA
21541 TCGAAACCGT GTACCTGGCG ACGCCCTTCT CCGCCGGCAA CGCCACAACA TAAAGAAGCA
21601 AGCAACATCA ACAACAGCTG CCGCATGGG CTCCAGTGAG CAGGAACCTG AAGCCATGTT
21661 CAAAGATCTT GGTGTGGGCG CATATTTTTT GGGCAGCTAT GACAAGCGCT TTCCAGGCTT
21721 TGTTCCTCCA CACAAGCTCG CCTGCGCAT AGTCAATACG CCGGTGGCG AGACTGGGG
21781 CGTACACTGG ATGCGCTTGT CCGTGAACCC GCACTCAAAA ACATGCTACC TCTTTAGGCC
21841 CTTTGGCTTT TCTGACAGC GACTCAAGCA GGTTTACAG TTTAGTACG AGTCACTCCT
21901 GCGCGTAGC GCCATTGCTT CTTCGCCGA CCGCTGTATA ACGCTGGAAG AGTCCACCCA
21961 AAGCGTAGC GGGCCCAACT CCGCGCGCTG TGGACTATTC TGCTGATGT TTCTCCAGGC
22021 CTTTGGCAAC TGGCCCAAAA CTCCCATGGA TCACAACCCC ACCATGAACC TTATTACGGG
22081 GGTACCCCAAC TCCATGCTCA ACAGTCCCCA GGTACAGCCC ACCCTGCGTC GCAACCAAGG
22141 ACAGCTCTAC AGCTTCTGG AGCGCCACTC GCGCTACTTC CCGAGCCACA GTGCGCAGAT
22201 TAGGAGCGCC ACTTCTTTTT GTCACTTGAA AAACATGTAA AAATAATGTA CTAGAGACAC
22261 TTTCAATAAA GGCAATGCT TTTATTGTA CACTCTGGGG TGATTATTTA CCCCCACCT
22321 TGCGTCTGC GCCCTTTAAA AATCAAAGGG GTTCTGCGCG GCATCGCTAT GCGCCACTGG
22381 CAGGACACAG TTGCGATACT GGTCTTAGT GCTCCACTTA AACTCAGGCA CAACCATCGG
22441 CCGCAGCTCG GTGAAGTTTT CACTCCACAG GCTGCGCACC ATCAACCAAG CTTTACGAG
22501 GTGCGGCGCC GATATCTTGA AGTCGCAGT GGGGCTTCCG CCGTGGCGCG GCGAGTTGGG
22561 ATACACAGGG TTGCAGCACT GGAACACTAT CAGCGCGGGG TGGTGACGCG TGSCCAGCAC
22621 GCTCTTTGCG GAGATCAGAT CCGCGTCCAG GTCTCGCGCG TTGCTCAGGG CCAACCGAGT
22681 CAACITTTGG AGCTGCTTTC CCAAAAAGGG CGCGTGCCCA GCGCTTGGAG TGCCTGCA
22741 CCGTAGTGCG ATCAAAGGT GACCGTGGCC GGTCTGGGCG TTAGGATACA GCGCTGCA
22801 AAAAGCCTTG ATCTGCTTAA AAGCCACCTG AGCCTTTGCG CTTTCAGAGA AGAACATGCC
22861 GCAAGACTTG CCGGAAACT GATTGCGCG ACAGCGCGCG TCGTGACGCG AGCACCTTGC
22921 GTCGGTGTG GAGATCTGCA CCACATTCG GCGCCACCGG TTTCTCAGA TCTTGGCCTT

FIGURE 23
(SHEET 7)

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22981 GCTAGACTGC TCCTTCAGCG CGGCTGCCC GTTTTCGCTC GTCACATCCA TTTCATCAC
23041 GTGCTCCTTA TTTATCATAA TGCTTCGCTG TAGACACTTA AGCTCGCCTT CGATCTCAGC
23101 GCAGCGGTGC AGCCACAACG CGCAGCCCGT GGGCTCGTGA TGCTGTAGAG TCACCTCTGC
23161 AAACGACTGC AGGTACGCGT GCAGGAATCG CCCCATCATC GTACAAAGG TCTTGTGCT
23221 GGTGAAGGTC AGCTGCAACC CGGGTGCTC CTGGTTGAGC CAGGTCTTGC ATACGGCCGC
23281 CAGAGCTTCC ACTTGTTCAG GCAGTAGTTT GAAGTTGCCC TTTAGATCGT TATCCACGTG
23341 GTACTTGTCC ATCAGCGCGC GGCAGGCTC CATGCCCTTC TCCACGCGAG ACACGATCGG
23401 CACACTCAGC GGGTTTCATCA CGGTAAATTC ACTTTCCGCT TCGCTGGGCT CTCTCTCTC
23461 CTCTTCGCTC CGCATACCA CCGCCACTGG GTGCTCTTCA TTCAGCCGCC GCACGTGCGG
23521 CTTACCTCCT TTGCCATGCT TGATTAGCAC CGTGGGTTG CTGAAACCCA CCATTGTGAG
23581 CGCCACATCT TCTCTTCTT CTCTGCTGTC CAGGATTACC TCTGGTGTG GCGGGCGCTC
23641 GGGCTTGGGA GAAGGGCGCT TCTTTTCTT CTGGGCGCA ATGGCCAAAT CGCGCGCGA
23701 GGTGATGGC CGCGGCGTGG GTGTGCGCGG CACGAGCGCG TCTTGTGTG AGTCTTCTC
23761 GTCTTCGGAC TCGATACGCC GCCTCATCCG CTTTTTGGG GCGCGCCGGG GAGGGCGCGG
23821 CGACGGGAGC GGGGACGACA CGTCTCCAT GGTGGGGGA CGTGGCGCG CACCGCGTCC
23881 GCGCTCGGGG GTGTTTCG CGTGCTCTC TTCCGACTG GCCATTTCT TCTCTATAG
23941 GCAGAAAGAG ATCATGGAGT CAGTCGAGAA GAAGGACAGC CTAACCCGCC CCTCTGAGTT
24001 CGCCACCACC GCCTCCACC ATGCCGCCAA CGCGCCTACC ACCTTCCCGG TCGAGGCACC
24061 CCGCTTGGAG GAGGAGGAAG TGATTATCGA GCAGGACCCA GGTTTTGTAA CGGAAGACGA
24121 CAGGAGCCGC TCAGTACCAA CAGAGGATAA AAGCAAGAC CAGGACAAAG CAGAGGCAA
24181 CGAGGAACAA GTCGGGCGGG GGGACGAAAG GCATGGCGAC TACCTAGATG TGGAGACGA
24241 CGTGCTGTG AAGCATCTGC AGCGCCAGTG CGCCATTATC TGCGACGCGT TGCAGAGCGG
24301 CAGCGATGTG CCGCTCGCCA TAGCGGATGT CAGCCTTGCC TACGAACGCC ACCTATCTC
24361 ACCGCGGTA CCGCCCAAAC GCGAGAAAG CGGCACATGC GAGCCCAACC CGCGCTCAA
24421 CTTCTACCCC GTATTTCGG TGCCAGAGGT GCTTGCCACC TATCACATCT TTTTCCAAA
24481 CTGCAAGATA CCGCTATCCT GCGGTGCCAA CCGCAGCGGA GCGGACAGC AGCTGGCCTT
24541 GCGCGAGGCG GTGTCATAC CTGATATGCG CTGCTCAAC GAAGTGCCAA AATCTTTGA
24601 GGTCTTGTGA CGCGACGAGA AGCGCGCGCG AAACGCTCTG CAACAGSAAA ACAGCGAAAA
24661 TGAAGATCAC TCTGGAGTGT TGGTGGAACT CGAGGGTGAC AACGCGCGCC TAGCGTACT
24721 AAAACGCGAG ATCGAGGTCA CCGACTTTGC CTACCGGCGA CTTAACCTAC CCGCCAGGT
24781 CATGAGCACA GTCATGAGTG AGCTGATCGT GCGCGTGCG CAGCCCTGCG AGAGGGATGC
24841 AAATTTGCAA GAACAAACAG AGGAGGSCCT ACCCGCAGTT GCGGACGAGC AGCTAGCGCG
24901 CTGGCTTCAA ACCGCGSAGC CTGCCGACTT GGAGGAGGSA CGCAACTAA TGTGCGCGC
24961 AGTGCTCGT ACCGTGGAGC TTGAGTGATC GCAGCGGTTT TTTGCTGACC CGGAGATGCA
25021 GCGCAAGCTA GAGGAAACAT TGCACTACAC CTTTCGACAG GGTCTAGTAC GCCAGGCTG
25081 CAAGATCTCC AACGTGGAGC TCTGCAACCT GGTCTCCTAC CTTGGAATTT TGCAOGAAAA
25141 CCGCCTTGGG CAAACGTTGC TTCATTCCAC GCTCAAGGGC GAGGCGCGCC GCGACTACGT
25201 CCGGACTGCG GTTTACTTAT TTCTATGCTA CACTGGCGAG ACGGCCATGG GCGTTGGCA
25261 CGAGTGCTTG GAGGAGTGCA ACCTCAAGGA GCTGCAGAAA CTGTAAAGC AAAACTTGAA
25321 GACCTATGCG ACGGCTTCA ACGAGCGCTC CGTGCGCGCG CACCTGGCGG ACATCATTTT
25381 CCGGACACCG CTGCTTAAAA CCGTGCAACA GGGTCTGCCA GACTTCACCA GTCAAGCAT
25441 GTTGCAAGAA TTAGGAACCT TTATCCTAGA GCGCTCAGGA ATCTTGCCCG CCACCTGCTG
25501 TGCACTTCTT AGCGACTTTG TGCCCAATTA GTACCGCGAA TGCCCTCGCG CGCTTTGGGG
25561 CCACTGCTAC CTTCTGCAGC TAGCCAACTA CTTTGCTTAC CACTCTGACA TAATGGAAGA
25621 CGTGAGCGGT GACGGTCTAC TGGAGTGTCA CTGTGCTGCG AACCTATGCA CCGCGCACCG
25681 CTCCCTGGTT TGCAATTCGC AGCTGCTTAA CGAAAGTCAA ATTATCGGTA CCTTTGAGCT
25741 GCAGGGTCCC TGCGCTGAGC AAAAGTCCCG GGCTCGGGGG TTGAACTCA CTCGCGGCT
25801 GTGACGCTCG GCTTACCTTC GCAAAATTGT ACCTGAGGAC TACCAGCGCC ACAGAGTTAG
25861 GTTCTACGAA GACCAATCCC GCGCGCCAAA TCGGGAGCTT ACCCGCTGCG TCATTACCCA
25921 GGGCCACATT CTTGGCAAT TGCAAGCCAT CAACAAAGCC CGCCAAAGT TCTGTACTCG
25981 AAAGGGACCG GGGGTTTACT TGGACCCCA GTCCGCGAG GAGCTCAACC CAATCCCCC
26041 GCGCGCGCAG CCGTATCAGC AGCAGCCCGG GCGCTTGTCT TCCAGGATG GCACCCAAAA
26101 AGAAGCTGCA GCTGCGCGCG CCACCCACGG ACGAGGAGGA ATACTGGGAC AGTCAGGCGG
26161 AGGAGGTTTT GAGCGAGGAG GAGGAGGACA TGATGGAAGA CTGGGAGAGC CTAGACGAGG
26221 AAGCTTCCGA GGTGGAAGAG GTGTGAGAGC AAACACCGTC ACCCTCGGTC GCATTCCCTT
26281 CCGCGCGCGC CCAAAATCG GCACCGGTT CAGCATGCG TACAACTCC GCTCTCAGG
26341 CCGCGCGCGC ACTGCGCTT CCGGACCCCA ACCGTAGATG GGAACCACT GGAACGAGG

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FIGURE 23
(SHEET 8)

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26401 CCGGTAAGTC CAAGCAGCCG CCGCCGTTAG CCCAAGAGCA ACAACAGCGC CAAGGCTACC
 26461 GCTCATGGCG CGGGCACAAG AACGCCATAG TTGCTTGCTT GCAAGACTGT GGGGGCAACA
 26521 TCTCCTTCGC CGCCCGCTTT CTTCTCTACC ATCAGCGCGT GGCCTTCCCC CGTAACATCC
 26581 TGCATTACTA CCGTCATCTC TACAGCCCAT ACTGCACCGG CGGCAGCGGC AGCGGCAGCA
 26641 ACAGCAGCGG CCACACAGAA GCAAGGCGGA CCGGATAGCA AGACTCTGAC AAAGCCCAAG
 26701 AAATCCACAG CGCGGCGAGC AGCAGGAGGA GGAGCGCTGC GTCTGGCGCC CAACGAACCC
 26761 GTATCGACCC GCGAGCTTAG AAACAGGATT TTTCCCACTC TGTATGCTAT ATTTCAACAG
 26821 AGCAGGGGCC AAGAACAAGA GCTGAAAATA AAAACAGGTT CTCTGCGATC CCTCACCCGC
 26881 AGCTGCTGTG ATCACAAGAAG CGAAGATCAG CTTGCGCGCA CGCTGGAAAG CCGGAGGGCT
 26941 CTCTTCAGTA AATACTGCGC GCTGACTCTT AAGGACTAGT TTCGCGCCCT TTCTCAAAAT
 27001 TAAGCGCGAA AACTACGTCA TCTCCAGCGG CCACACCCGG CGCCAGCACC TGTGTCAGC
 27061 GCCATTATGA GCAAGGAAT TCCACGCCCC TACATGTGGA GTTACCAGCC ACAAAATGGGA
 27121 CTTGCGGCTG GAGCTGCCCA AGACTACTCA ACCCGAATAA ACTACATGAG CGCGGGACCC
 27181 CACATGATAT CCGCGGTCAA CGGAATCCGC GCCCACCGAA ACCGAATCTT CTGTGAACAG
 27241 GCGGCTATTA CCACCAACCC TCGTAATAAC CTTAATCCCC TTAGTTGGCC CGCTGCCCTG
 27301 GTGTACCAAG AAAGTCCCGC TCCCACTACT GTGGTACTTC CCAGAGACGC CCAGGCGGAA
 27361 GTTCAGATGA CTAATCAGG GCGCGAGCTT GCGGCGCGGT TTCGTACAGG GGTGCGGTG
 27421 CCGGGGCGAG GTATAACTCA CCTGCAATC AGAGGGCGAG GTATTCACT CAACGACGAG
 27481 TCGGTAGCTT CCTCGCTTGG TCTCGTCCG GACGGGACAT TTCAGATCGG CGGCGCGCGC
 27541 CGTCTTCTAT TCAAGCCTCG TCAGGCAATC CTAATCTGCG AGACCTGTCG CTCTGAGCGC
 27601 CGCTCTGGAG GCATTGGAAC TCTGCAATTT ATTGAGGAGT TTTGTCCATC GGTCTACTTT
 27661 AACCCCTTCT CCGGACCTCC CCGCACTAT CCGGATCAAT TTATTCTTAA CTTTGAACGC
 27721 GTAAAGGACT CGCGGAGCGG CTACGACTGA ATGTTAAGTG GAGAGGCGAG GCAACTGCCG
 27781 CTGAACACCC TGGTCCACTG TCGCCGCGAC AAGTGCTTTG CCCGCGACTC CGGTGAGTTT
 27841 TGCCTTTTAT AATTGCGCGA GGATCATATC GAGGGCCCGG CGCACGCGGT CCGGCTTACC
 27901 GCCCAGGGAG AGCTTGCCCG TAGCCTGATT CCGGAGTTTA CCCAGCGCCC CTGTGATGTT
 27961 GAGCGGGACA GGGGACCCCT TGTCTCACT GTGATTGCA ACTGTCTTAA CTTTGGATT
 28021 CATCAAGATC TTTGTTGCCA TCTCTGTGCT GAGTATAATA AATACAGAAA TTAATAATA
 28081 CTGGGGCTCC TATCGCCATC CTGTAAACGC CACGCTCTTC ACCCGCCCAA GCAACCAAG
 28141 GCGAACCTTA CTTGGTACTT TTAACATCTC TCCCTCTGTG ATTTACAACA GTTTCAACCC
 28201 AGACGGAGTG AGTCTACGAG AGAACCTCTC CGAGCTCAGC TACTCCATCA GAAAAACAC
 28261 CACCTCCTTT ACCTGCGCGG AACGTACGAG TGGCTCACCG GCCGCTGCAC CACACCTACC
 28321 GCTGACCGT AAACCCAGCT TTTTCCGGAC AGACCTCAAT AACTCTGTCT ACCAGAACAG
 28381 GAGGTGAGCT TAGAAAAACC TTAGGGTATT AGGCCAAAGG CGCAGCTACT GTGGGGTTTA
 28441 TGAACAATTC AAGCAACTCT ACGGGCTATT CTAATTGAGG TTTCTCTAGA AGTCAGGCTT
 28501 CCTGGATGTC AGCATCTGAC TTTGGCCAGC ACCTGTCCCG CGGATTGTGT CCAATCCAAC
 28561 TACAGCGACC CACCCCTAAC GAGATGACCA ACACAACCAA CGCGGCGCGC GCTACCGGAC
 28621 TTACATCTAC CACAAATACA CCCCAAGTTT CTGCGCTTGT CAATAACTGG GATAACTTGG
 28681 GCATGTGGTG GTTCTCCATA GCGCTTATGT TTGTATGCTT TATTATTATG TGGCTCATCT
 28741 GCTGCTTAAA GCGCAACCGC GCCCGACCAC CCATCTATAG TCCCATCATT GTGCTACACC
 28801 CAACAATGTA TGGAAATCCAT AGATTGGACG GACTGAAACA CATGTTCTTT TCTCTTACAG
 28861 TATGATTAAA TGAGATCTAG AAATGGACGG AATTATTACA GAGCAGCGCC TGCTAGAAAG
 28921 ACGCAGGGCA CGGCGCGAGC AACAGCGCAT GAATCAAGAG CTCCAAGACA TGGTTAACTT
 28981 GCACCACTGC AAAAGGGGTA TCTTTGTCTT GGTAAAGCAG GCCAAGTCA CTTACGACAG
 29041 TAATACCACC GGACACCGCC TTAGCTACAA GTTGCCAACC AAGCGTCAGA AATTGGTGGT
 29101 CATGGTGGGA GAAAAGCCCA TTACCATAAC TCAGCACTCG GTAGAAACCG AAGGCTGCAT
 29161 TCACTCACTT TGTCAAGGAC CTGAGGATCT CTGCAACCTT ATTAAGACCC TGTGGGTCT
 29221 CAAAGATCTT ATTCCCTTTA ACTAATAAAA AAAAATAATA AAGCATCACT TACTTAAAT
 29281 CAGTTAGCAA ATTTCTGTCC AGTTTATTTA GCAGCACCTC CTTGCCCTCC TCCAGCTCT
 29341 GGTATTGACG CTTCTCTCTG GCTGCAAACT TTCTCCACAA CTTAAATGGA ATGTGAGTTT
 29401 CTTCTGTGTC CTGTCCATCC GCACCCACTA TCTTCATGTT GTTGCAGATG AAGCGGCGAA
 29461 GACCGTCTGA AGATACCTTC AACCCGCTGT ATCCATATGA CACGGAACCC GGTCTCCCAA
 29521 CTGTGCTTCT TCTTACTCTT CCGTTTGTAT CCCCCTATGG GTTTCAAGAG AGTCCCGCTG
 29581 GGGTACTCTC TTTGCGCCTA TCCGAACCTC TAGTTAGCTC CAATGGCATG CTTGCGCTCA
 29641 AAATGGGCAA CGGCTCTCTT CTGGACGAGG CCGGCAACCT TACCTCCCAA AATGTAAACA
 29701 CTGTGAGCCC ACCTCTCAAA AAAACCAAGT CAAACATAAA CCTGGAATAA TCTGCACCCC
 29761 TCACAGTTAC CTCAGAAGCC CTAACGTGGG CTGCGCGCGC ACCTCTAATG GTGCGGCGCA

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FIGURE 23
(SHEET 9)

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29821 ACACACTCAC CATGCAATCA CAGGCCCGGC TAACCGTGCA CGACTCCAAA CTTAGCATTG
 29881 CCACCCCAAGG ACCCCCTCACA GTGTGAGAAG GAAAGCTAGC CCTGCAAAACA TCAGGCCCGCC
 29941 TCACCACCAC CGATAGCAGT ACCCTTACTA TCAGTGCCTC ACCCCCTCTA ACTACTGCCA
 30001 CTGGTAGCTT GGGCATTGAC TTGAAAGAGC CCATTITATC ACAAATGGA AAAGTGGAC
 30061 TAAAGTACGG GGTCTCTTTG CATGTAACAG ACGACCTAAA CACTTTGACC GTAGCAACTG
 30121 GTCCAGGTGT GACTATTAAAT AATACTTCCT TGCAAACTAA AGTTACTGGA GCCTTGGGTT
 30181 TTGATTCCAA AGGCAATATG CAACTTAATG TAGCAGGAGG ACTAAGGATT GATTCTCAAA
 30241 ACAGACGCCCT TATACTTGAT GTTAGTTATC GGTITGATGC TCAAAACCAA CTAAATCTAA
 30301 GACTAGGACA GGGCCCTCTT TTTATAAACT CAGCCCACAA CTITGGATTAT AACTACAACA
 30361 AAGGCCCTTA CTTGTTTACA GCTTCAAAAC ATTCCAAAAA GCTTGAGGTT AACCTAAGCA
 30421 CTGCCAAGGG GTTGATGTTT GACGCTACAG CCATAGCCAT TAATGCAGGA GATGGGCTTG
 30481 AATTGGTTTC ACCTAATGCA CCAACACAA ATCCCTCAA AACAAAAAT GGCCATGGCC
 30541 TAGAATTGTA TTCAAACAG GCTATGGTTC CTAACTAGG AACTGGCCTT AGTTTGGACA
 30601 GCACAGGTGC CATTACAGTA GGAAACAAA ATAATGATAA GCTAATCTTG TGGACCACAC
 30661 CAGCTCCATC TCCTAACTGT AGACTAAATG CAGAGAAAGA TGCTAAACTC ACTTTGGTCT
 30721 TAACAAAATG TGGCAGTCAA ATACTTGCTA CAGTTTCAGT TTTGGCTGTT AAAGGCAGTT
 30781 TGGCTCCAAAT ATCTGGAGCA GTTCAAAAGT CTCTACTTAT TATAAGATTT GACGAAAATG
 30841 GAGTGTACTT AAACAATTC TCTCTGGACC CAGAATATTT GAACTTTAGA AATGGAGATC
 30901 TTACTGAAGG CACAGCCTAT ACAAACGCTG TTGGATTAT GCCTAACCTA TCAGCTTATC
 30961 CAAAATCTCA CGGTAAAACT GCCAAAAGTA ACATTGTGAG TCAAGTTTAC TTAACCGGAG
 31021 ACAAACCTAA ACCTGTAAAC CTAACCATTA CACTAAACGG TACACAGGAA ACAGGAGACA
 31081 CACTCCCAAG TGCATACTCT ATGTTCATTT CATGGGACTG GTCTGGCCAC AACTACATTA
 31141 ATGAAATATT TGCCACATCC TCTTACACTT TTTACATCAT TGCCCAAGAA TAAAGAAATG
 31201 TTTGTGTTAT GTTTCACAGT GTTTATTTT CAATTGCAGA AAATTTCAAG TCATTTTCA
 31261 TTCAAGTAGT TAGCCCAACC ACCACATAGC TTATACAGAT CACCGTACCT TAATCAAACT
 31321 CACAGAACCC TAGTATTCAA CCGTCCACCT CCTTCCCAAC ACACAGAGTA CACAGTCTCT
 31381 TCTCCCGGCG TGCCCTTAAA AAGCATATA TCATGGGTAA CAGACATATT CTTAGGTGTT
 31441 ATATTCCACA CGGTTTCTCTG TCGAGCCAAA CGCTCATCAG TGATATTAAAT AAACCTCCCG
 31501 GGCAGCTCAC TTAAGTTTAT GTCCGTCTCC AGCTGCTGAG CCACAGGCTG CTGTCCAATC
 31561 TGGGTTTGTG TAACGGGGCGG CGAAGGAGAA GTCCAGGCTC ACATGGGGGT AGAGTCAATA
 31621 TCGTGTCTCA GGATAGGGCG GTGGTGTCTG AGCAGGCGCG GAATAAACTG CTGCGCGCGC
 31681 CGCTCCGTCG TGCAAGGAATA CAACATGGCA GTGGTCTCCT CAGCGATGAT TCGCACCGCC
 31741 CGCAGCATAA GGCCTCTGT CCTCGGGCA CAGCAGCGCA CCTGATCTC ACTTAAATCA
 31801 GCACAGTAAC TGCAAGCAG CACCAATAA TTGTTCAAAA TCCACAGTG CAAGGCGCTG
 31861 TATCCAAAGC TCATGGCGGG GACCACAGAA CCCACGTGGC CATCATCCA CAAGCGCAGG
 31921 TAGATTAAAT GGCAGCCCTC CATAAACAGC CTGGACATAA ACATTACCTC TTTTGGCATG
 31981 TTGTAATTCA CCACCTCCCG GTACCATATA AACCTCTGAT TAAACATGGC GCCATCCACC
 32041 ACCATCTTAA ACCAGCTGGC CAAAACCTGC CCGCCGGCTA TACACTGCAG GGAACCGGGA
 32101 CTGGAACAAT GACAGTGGAG AGCCAGGAC TCCTAACCAT GGATCATCAT GCTCGTCATG
 32161 ATATCAATGT TGGCACAACA CAGGCACAGC TGCTATACCT TCCTCAGGAT TACAGCTCC
 32221 TCCCGCGTTA GAACCATATC CCAGGGAACA ACCCATTCCT GAATCAGGCT AAATCCCA
 32281 CTGCAAGGAA GACCTCGCAC GTAACTCAGC TTGTGCTATG TCAAGTGTG ACATTGGGGC
 32341 AGCAGCGGAT GATCCTCCAG TATGTAGCG CGGTTTCTG TCTCAAAAGG AGGTAGACGA
 32401 TCCCTACTGT ACGGAGTGCG CCGAGACAAC CGAGATCGTG TTGTCGTGAT TGTCTAGCA
 32461 AATGGAACGC CGGACGTAGT CATATTTCTT GAAGCAAAAC CAGGTGCGGG CGTGACAAAC
 32521 AGATCTGCGT CTCGGTCTC GCGGCTTAGA TCGCTCTGTG TAGTAGTTGT AGTATATCCA
 32581 CTCTCTCAA GCATCCAGGC GCCCCTTGGC TTGGGTTCT ATGTAAACTC CTTCTAGCGC
 32641 CGCTGCGCTG ATAACATCCA CCACCGCAGA ATAAGCCACA CCCAGCCAAC CTACACATTC
 32701 GTTCTGCGAG TCACACACGG GAGGAGCGGG AAGAGCTGTA AGAACCATGT TTTTTTTTTT
 32761 ATTCCAAAAG ATTATCCAAA ACCTCAAAAT GAAGATCTAT TAAAGTGAACG CGCTCCCTC
 32821 CGGTGGCGTG GTCAAACTCT ACAGCCAAAG AACAGATAAT GGCATTGTGA AGATGTTGCA
 32881 CAATGGCTTC CAAAAGGCAA ACGGCCCTCA CGTCCAGTG GACGTAAAGG CTAACCCCTT
 32941 CAGGGTGAAT CTCTCTTATA AACATTCCAG CACCTTCAAC CATGCCCAA TAATTCTCAT
 33001 CTGCGCACCT TCTCAATATA TCTTAAGCA AATCCCGAAT ATTAAGTCCG GCCATTGTAA
 33061 AAATCTGCTC CAGAGCGCCC TCCACCTTCA GCTCAAGCA GCGAATCATG ATTGCAAAAA
 33121 TTCAGGTTCC TCACAGACCT GTATAAGATT CAAAAGCGGA ACATTAAACA AAATACCGCG
 33181 ATCCGTAGG TCCCTTCGCA GGGCCAGCTG AACATAATCG TGCAAGTCTG CACGAGCCAG

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FIGURE 23
(SHEET 10)

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33241 CGCGGCCACT TCCCCGCCAG GAACCTTGAC AAAAGAACCC ACACGTGATTA TGACACGCAT
33301 ACTCGGAGCT ATGCTAACCA GCGTAGCCCC GATGTAAGCT TTGTTGCATG GCGGCGGATA
33361 TAAATGCAA GGTGCTGCTC AAAAAATCAG GCAAGCCTC GCGCAAAAAA GAAAGCACAT
33421 CGTAGTTCATG CTCATGCAGA TAAAGGCAGG TAAGCTCCGG AACCACCACA GAAAAAGACA
33481 CCATTTTTCT CTCAAACATG TCTGCGGGTT TCTGCATAAA CACAAATAA AATAACAAAA
33541 AAACATTAA ACATTAGAAG CCTGTCTTAC AACAGGAAAA ACAACCCTTA TAAGCATAAG
33601 ACGGACTACG GCCATGCCGG CGTGACCGTA AAAAACTGG TCACCGTGAT TAAAAAGCAC
33661 CACCGACAGC TCCTCGGTCA TGTCGGGAGT CATAATGTAA GACTCGGTAA ACACATCAGG
33721 TTGATTTCATC GGTCACTGCT AAAAAAGCAC CGAAATAGCC CGGGGGAATA CATACCCGCA
33781 GCGGTAGAGA CAACATTACA GCCCCCATAG GAGGTATAAC AAAATTAATA GGAGAGAAAA
33841 ACACATAAAC ACCTGAAAAA CCTCCTGCC TAGGCAAAAT AGCACCCCTCC CGCTCCAGAA
33901 CAACATACAG CGCTTCACAG CGGCAGCCTA ACAGTCAGCC TTACCCGTAA AAAAGAAAAAC
33961 CTATTAAAAA AACACCACTC GACACGGCAC CAGCTCAATC AGTCACAGTG TAAAAAGGG
34021 CCAAGTGCAG AGCGAGTATA TATAGGACTA AAAAATGACG TAACGGTTAA AGTCCACAAA
34081 AAACACCCAG AAAACCGCAC GCGAACCTAC GCCCAGAAAC GAAAGCCAAA AAACCCACAA
34141 CTTCTCAAA TCGTCACTTC CGTTTTCCCA CGTTACGTAA CTTCCCATTT TAAGAAAACT
34201 ACAATTCCCA ACACATACAA GTTACTCCGC CCTAAAACCT ACGTCACCCG CCCCCTTCCC
34261 ACGCCCCGCG CCACGTACA AACTCCACCC CCTCATTATC ATATTGGCTT CAATCCAAAA
34321 TAAGGTATAT TATTGATGAT G

//

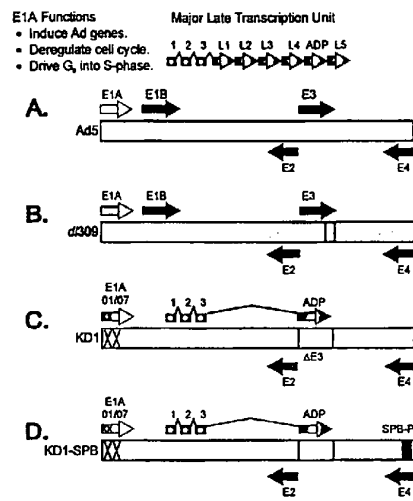


FIGURE 24

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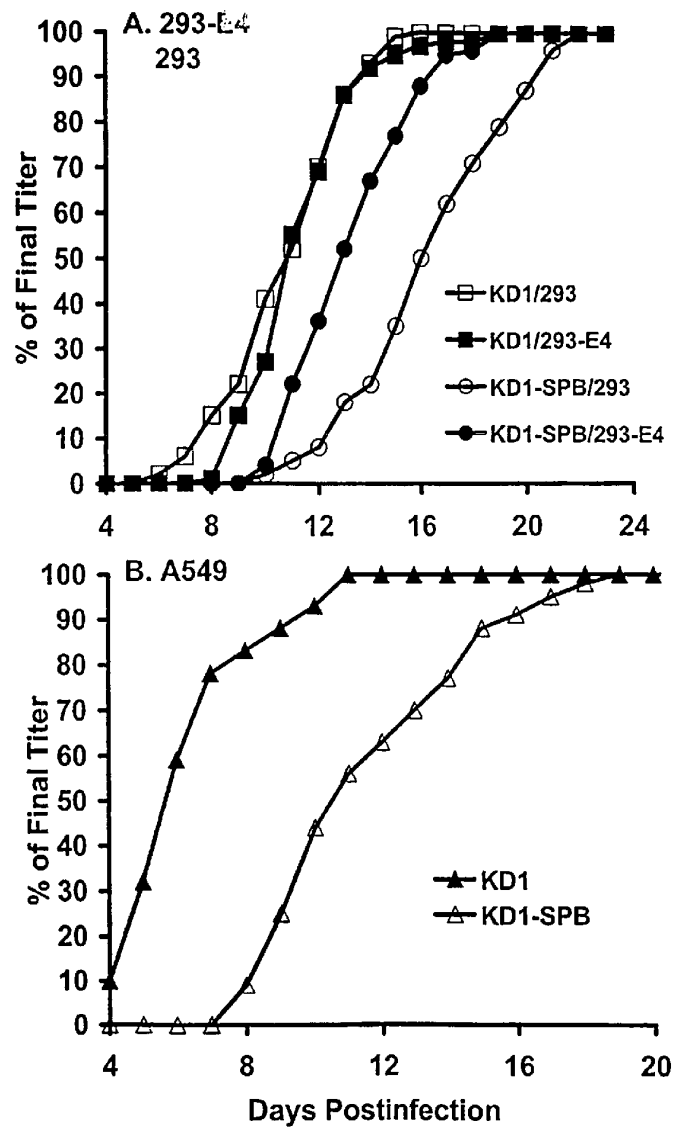


FIGURE 25

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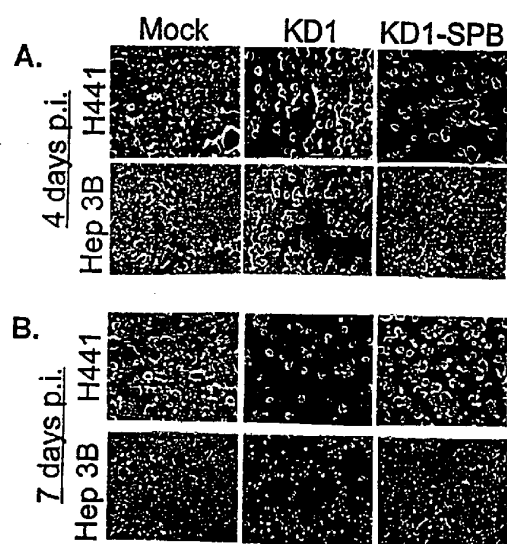


FIGURE 26

S8/66

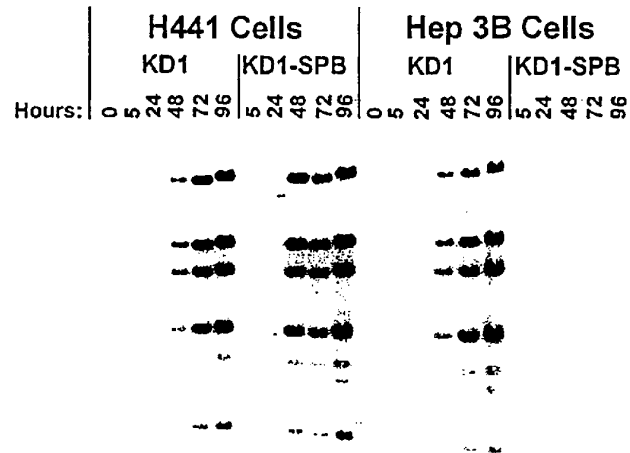


FIGURE 27A

59 | 66

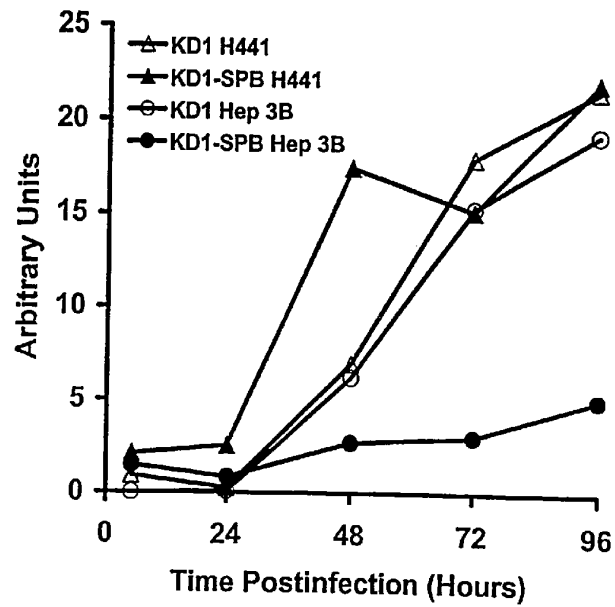


FIGURE 27B

60 | 66

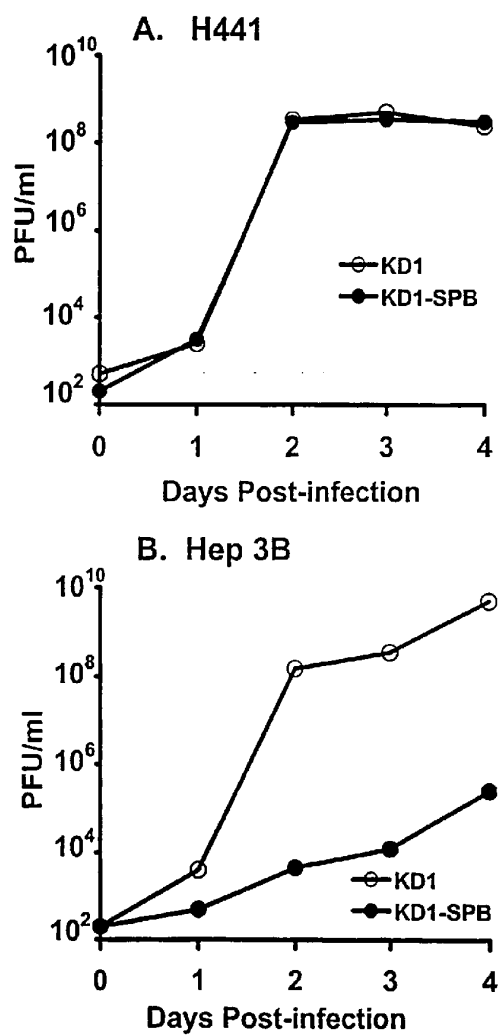


FIGURE 28

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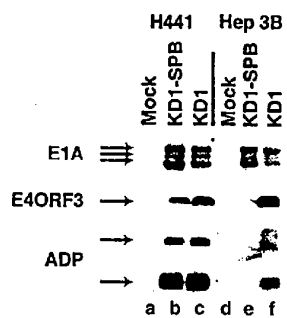


FIGURE 29

62|66

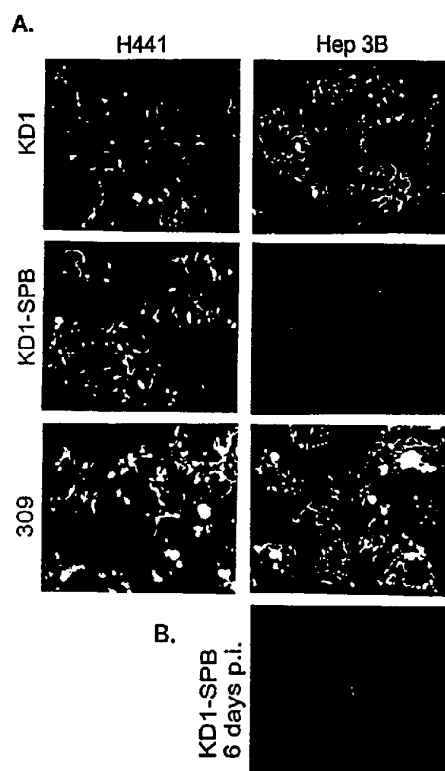


FIGURE 30

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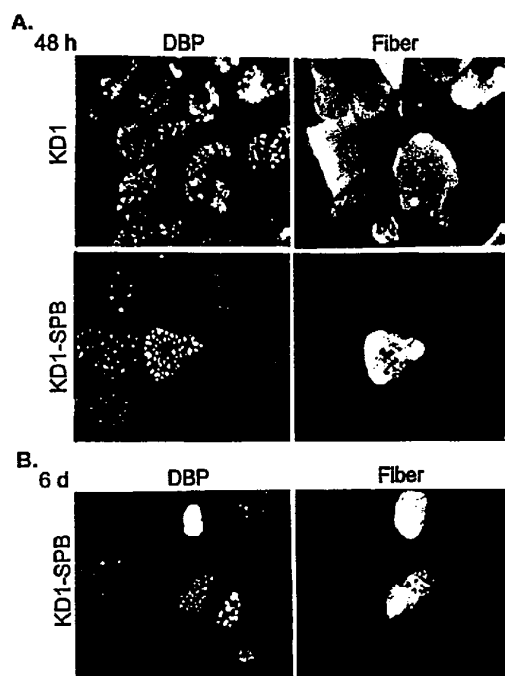


FIGURE 31

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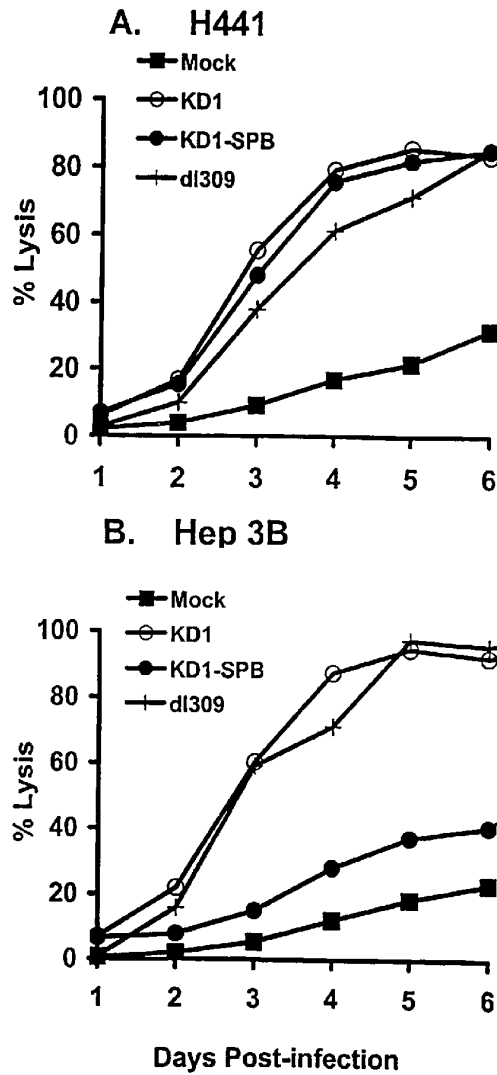


FIGURE 32

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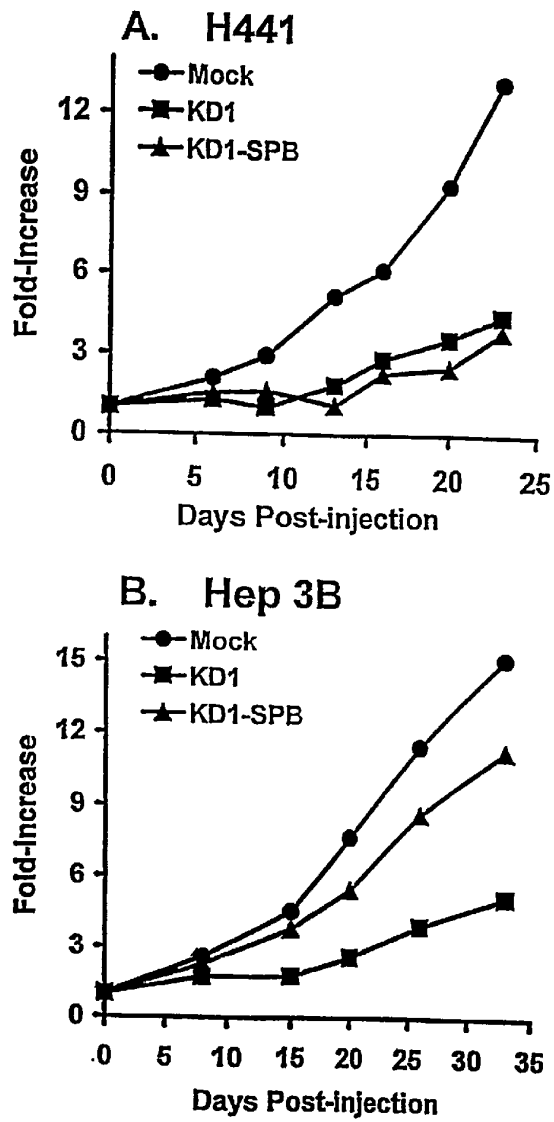


FIGURE 33

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