Fatty Acid Synthase Dimers Containing Catalytically Active β-Ketoacyl Synthase or Malonyl/Acetyltransferase Domains in Only One Subunit Can Support Fatty Acid Synthesis at the Acyl Carrier Protein Domains of Both Subunits*

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A double-tagging, dual affinity chromatographic procedure, which permits isolation of dimers independently mutated in each subunit, has been exploited to probe the functional topology of the animal fatty acid synthase. Dimers were engineered in which the chain-terminating thioesterase reaction was compromised by mutation of the (active-site) serine residue in both subunits; these dimers assembled two long-chain fatty acyl moieties, which remained covalently linked to the 4′-phosphopantetheine residues of the two acyl carrier protein domains. Significantly, dimers that contained an additional mutation that compromised the activity of either the β-ketoacyl synthase or malonyl/acetyltransferase activity in only one subunit also assembled two long-chain acyl moieties. In contrast, in a control experiment, introduction of an additional mutation that compromised the function of the acyl carrier protein domain in only one subunit resulted in the assembly of only one long-chain acyl moiety per dimer. Because the β-ketoacyl synthase and malonyl/acetyltransferase domains are located near the amino terminus of the polypeptide and the acyl carrier protein domain near the carboxyl terminus, these results support a modified model for the animal fatty acid synthase in which head-to-tail functional contacts are possible both within as well as between subunits.

The animal FAS1 consists of two identical polypeptides, each carrying six enzymes and an acyl carrier protein, that are juxtaposed to form two centers for the synthesis of palmitic acid from acetyl- and malonyl-CoA. The six catalytic domains are clustered in two regions separated by ~600 residues that have not been ascribed a functional role. Thus the β-ketoacyl synthase, malonyl/acetyltransferase, and dehydrase domains are located within the amino-terminal half of the polypeptide, whereas the enoyl reductase, β-ketoacyl reductase, and thioesterase are located in the carboxyl-terminal half; the ACP domain, which carries the 4′-phosphopantetheine prosthetic group, is located between the β-ketoacyl reductase and thioesterase domains. The development of a model for the FAS was strongly influenced by two key observations. First, the monomeric form of the protein is inactive in the overall FAS reaction (1–3), yet two centers for palmitate synthesis are created by the association of subunits in the dimeric form (4, 5). Second, the two subunits can be cross-linked via the β-ketoacyl synthase, active-site, cysteine thiol of one subunit and the ACP 4′-phosphopantetheine thiol of the companion subunit (6). Thus, in the generally accepted model for the FAS, the two polypeptides lie side-by-side in a fully extended, antiparallel configuration such that each of the two centers for palmitate synthesis requires cooperation between catalytic domains located in the amino-terminal half of one subunit with those located in the carboxyl-terminal half of the adjacent subunit (7–9). Recently we have used a mutant complementation strategy to test and refine this model. The approach requires the engineering of various modified FASs, in which the activity of one of the functional domains is specifically compromised by mutation. Heterodimers formed from subunits containing different, single mutations may be capable of fatty acid synthesis if the two mutations are located on domains that normally cooperate with each other across the subunit interface (10). Application of this strategy has confirmed several features of the previously proposed model. For example, mutations in either the β-ketoacyl synthase or malonyl/acetyltransferase domain complement mutations in either the ACP or thioesterase domains, whereas mutations in the ACP and thioesterase domains fail to complement each other (11, 12). These findings confirmed that the β-ketoacyl synthase and malonyl/acetyltransferase domains, located in the amino-terminal half of the FAS polypeptide, can contribute to the same center of palmitate synthesis, as do the ACP and thioesterase domains of the companion subunit. However, additional complementation data revealed two features of the FAS topology that were not predicted by the model.

First, FAS compromised by a mutation in the dehydrase domain did not complement FASs containing a critical mutation in either the ACP or thioesterase domain but instead complemented FASs mutated in either the β-ketoacyl synthase or malonyl/acetyltransferase domain (11, 12). Thus, the dehydrase domain appears to belong to the carboxyl-terminal complementation group, even though it is physically located in the amino-terminal half of the polypeptide chain (13). The implication of this result is that the β-hydroxyacyl-ACP is converted to enoyl-ACP through the action of the dehydrase domain on the same subunit, rather than on the adjacent subunit, as was predicted by the earlier model. Because the dehydrase and ACP domains are separated by >1100 residues, this finding provided the first experimental evidence indicating that the two constituent polypeptides are not simply positioned side-by-side in a fully extended rigid conformation but can coil...
such that functional contact is possible between domains distantly located on the same polypeptide.

Second, mutations in the two amino-terminal domains, the \( \beta \)-ketoacyl synthase and malonyl/acyetyltransferase, complement each other, despite the fact that they also complement mutations in the ACP and thioesterase domains, which are located at the carboxyl terminus (11, 12). This unexpected finding raised the possibility that the malonyl/acyetyltransferase domain may be able to deliver substrates to either of the two ACPs in the dimer and that the condensation reaction may be catalyzed by the cooperation of the \( \beta \)-ketoacyl synthase with either of the two ACP domains. On the basis of these results, we proposed a revised model for the FAS in which the substrate loading and condensation reactions can be catalyzed by cooperation between domains across the subunit interface or by cooperation of domains from only one of the subunits (12). Active FASs that are reconstituted from inactive mutants by complementation necessarily contain only one functional center for fatty acid synthesis per dimer. However, if the modified model is indeed valid, it follows that FAS dimers containing only one inactive \( \beta \)-ketoacyl synthase or malonyl/acyetyltransferase domain ought to be capable of synthesizing fatty acids on both of the ACP domains. The objective of the present study was to test this hypothesis.

**EXPERIMENTAL PROCEDURES**

**Materials—**Anti-FLAG M2 monoclonal antibody and anti-FLAG M2 affinity gel were purchased from Eastman Kodak Co., and anti-His \(_{\text{6}}\) monoclonal antibody was from CLONTECH Laboratories Inc. (Palo Alto, CA). Ni-NTA agarose was obtained from Qiagen Inc. (Santa Clarita, CA). FLAG octapeptide (DYKDDDDK) was synthesized at the PAN Facility (Beckman Center, Stanford University Medical Center). The sources of other materials have been detailed in previous publications (10–14).

**Construction of His \(_{\text{6}}\) - and FLAG-tagged FAS cDNAs—**Construction and expression of full-length wild-type rat FAS, and mutants thereof, defective in the thioesterase, S2302A (11), malonyl/acyetyltransferase, S581A (12), ACP, S2151A (10), and \( \beta \)-ketoacyl synthase, K326A (11), domains, has been described in detail previously. A second \( \beta \)-ketoacyl synthase mutant, C161A was constructed by first generating a mutated partial fragment by polymerase chain reaction amplification using primer set M161T3 (5'-TCAAAGGCCACATGCTGCTGGA-CACAGCGGCTTCTAAGC5'T, 5'-CACTAGAATTCCTTA-GGTGGTGGGTTGAATGC) and pFAS 74.20 (partial FAS cDNA in pcUBM20) as template; the boldface nucleotides encode alanine, glycine, or valine at residue 161. A more detailed account of these procedures has been published previously (15). Authenticity of the amplification product was confirmed by DNA sequencing, and the fragment was moved into the full-length wild-type FAS DNA.

Double mutants containing mutations in two different FAS domains were generated by replacing a section of cDNA in the parental single mutant construct with an appropriately mutated fragment. Successful introduction of the mutations was confirmed by standard techniques. The affinity tags hexahistidine (His \(_{\text{6}}\)), which permits purification by metal-ion chelation chromatography, and FLAG-tag (N'-DYKDDDDK), which permits purification by immunoaffinity chromatography, were incorporated either at the carboxyl terminus or between amino acid residues 1154 and 1157 of the FAS (15).

**Expression and Purification of Tagged FASs—**Sf9 cells were infected with purified recombinant viruses and cultured for 48–72 h at 27 °C, and the tagged FAS proteins were purified from the cytosols as described earlier (14); glyceral (10%, v/v) was included in all buffers used for chromatography. FAS was detected in chromatographic fractions by monitoring \( \beta \)-ketoacyl reductase activity.

**Randomization of FAS Subunits—**Typically, FLAG-tagged and His \(_{\text{6}}\)-tagged FASs (in 25–35 mM potassium phosphate buffer, pH 7.0, 1 mM dithiothreitol, 0.1 mM EDTA, and ~1% glyceral) were aged at 4 °C for 7–9 days to promote dissociation into their component subunits (1). Spontaneous reassociation of the subunits was induced by adjustment of the solvent to ~200 mM potassium phosphate, pH 7.0, and 10% glyceral and incubation at 30 °C for 75 min.

**Isolation of Heterodimers by Affinity Chromatography—**The subunits in a mixture of His \(_{\text{6}}\) - and FLAG-tagged dimers were randomized, as described above, and the heterodimers containing one His \(_{\text{6}}\)-tagged and one FLAG-tagged subunit were isolated by chromatography successively on an anti-FLAG column, using the FLAG octapeptide in the eluting buffer, and then on a Ni-NTA column, by including imidazole in the eluting buffer (15). Imidazole was removed from the FAS using a DEAE anion exchange cartridge (14).

**Removal of Endogenously Secreted Fatty Acyl Moieties From FAS Mutants Lackig Catalytically Active Thioesterase Domains—**All preparations of FAS that included the S2302A mutation in the thioesterase domain were treated with highly purified recombinant rat mammary gland thioesterase (18) to remove fatty acyl chains that had been synthesized in the insect cell host and that remained covalently linked to the prethiolated fatty acyl synthase. Typically, FAS preparations (5–11 \( \mu \)l) were treated with thioesterase II (1–2 \( \mu \)l) for 30 min at 37 °C, and the thioesterase was removed by affinity chromatography as described above.

**Analysis of Fatty Acyl Moieties Bound Cowently to FAS—**FASs were incubated with [\( ^{2-14}\)C]malonyl-CoA, acetyl-CoA, and NADPH; unused acetyl and malonyl moieties were unloaded from the FAS by incubation with CoASH (17); and the protein was collected by precipitation with ethanol. Covalently bound fatty acids were released from the protein by treatment with performic acid and derivatized with phenyl-8 (15). The phenacyl fatty acids were analyzed by high performance liquid chromatography as described earlier (18). The recovery of radioiodinated acyl moieties through the ethanol precipitation and performic acid treatment averaged 87%.

**Enzyme Assays—**Overall fatty acid synthesizing activity was measured spectrophotometrically (19). \( \beta \)-Ketoacyl reductase activity was monitored spectrophotometrically using trans-1-decenoate as a model substrate (14, 20). Malonyl/acyetyltransferase was assayed radiochemically using radiolabeled malonyl- or acetyl-CoA as acyl donor and pantetheine as acceptor; the product was separated from unreacted substrate by thin layer chromatography (21). The interthiol acyltransferase activity associated with the \( \beta \)-ketoacyl synthase domain was assayed using decanoyl-S-pantetheine as acyl donor and CoASH as acceptor (22). A unit of activity is defined as 1 \( \mu \)mol of substrate (NADPH in the case of the overall FAS reaction) used/min.

**SDS-Polyacrylamide Gel Electrophoresis and Western Analysis—**FAS preparations were analyzed by SDS-polyacrylamide gel electrophoresis, using 7.5% polyacrylamide gels, and by Western analysis using either murine anti-FLAG M2 monoclonal antibody (10 \( \mu \)g/ml) or murine anti-His \(_{\text{6}}\) monoclonal antibody (1:2000 dilution) as the primary antibody and alkaline phosphatase-coupled goat anti-mouse IgG antibody as the secondary antibody (15).

**RESULTS AND DISCUSSION**

**Overall Strategy—**The objective of this study was to construct FAS heterodimers in which either the malonyl/acyetyltransferase or \( \beta \)-ketoacyl synthase activity of only one subunit was compromised by mutation and then to determine whether one or both ACPs were able to participate in fatty acid production. The heterodimers were formed by randomization of the subunits from two different homodimeric FAS species. To isolate a heterodimer population free of contaminating parental homodimers, we made use of a recently described double-tagging procedure in which each of the parental homodimers is tagged with a different affinity label, either hexahistidine or FLAG tag; the heterodimeric species can then be purified by a double affinity chromatographic procedure (15), because each of the tagged homodimeric species binds to only one of the two affinity matrices, whereas the doubly tagged heterodimers bind to both. Two experimental systems were explored. First, to facilitate analysis of the number of fatty acyl chains assembled by such heterodimers, we mutated the active-site serine residue 2302 of the thioesterase domain to alanine in both parental homodimers. This mutation compromises the ability of the FAS to release the fatty acyl chain formed in the thioesterase domain (11) so that, by using radiolabeled acetyl-CoA as substrate, the stoichiometry of fatty acyl chain assembly can be ascertained (23). Thus, the thioesterase mutation (TE\(^{\text{A}}\)) was combined with either malonyl/acyetyltransferase, S581A (MAT\(^{\text{A}}\)), or \( \beta \)-ketoacyl synthase, K326A (KSY\(^{\text{A}}\)), in the parental homodimers. Ser-581 is the nucleophile for the malonyl/acyetyltransferase reaction (12), and Lys-326 plays an essential role in...
Heterodimers were formed by randomization of subunits from pairs of parental homodimers and isolated by dual column affinity chromatography. WT, wild type. Data represent means ± S.D.s for triplicate determinations.

The β-ketoacyl synthase reaction (22). The amino acid substitutions in the malonyl/acyltransferase and β-ketoacyl synthase domains affect only the activity of the mutated domain (10, 12). Second, to assess the rate of fatty acid synthesis by FASs containing only one active malonyl/acyltransferase or β-ketoacyl synthase domain, we constructed parental homodimers carrying either the malonyl/acyltransferase or β-ketoacyl synthase mutations in the context of normal thioesterase domains. In the β-ketoacyl synthase mutation used for these experiments, the Cys-161 nucleophile was replaced by Ala. The activity of the heterodimers formed by hybridizing these homodimers with wild-type homodimers was assessed by normal steady-state kinetic analysis. As a control, in both the determination of the stoichiometry of long acyl chain assembly and in the steady-state kinetic experiments, we also introduced a mutation, S2302A, that eliminates posttranslational insertion of the 4′-phosphopantetheine prosthetic group in the ACP domain (10).

Characterization of Heterodimers Containing Only a Single Catalytically Active Malonyl/acyltransferase or β-Ketoacyl Synthase Domain—To confirm the authenticity of the various heterodimer preparations used in the study, we assayed both the heterodimers and their parental homodimers for malonyl/acyltransferase and β-ketoacyl synthase activity (Tables I and II, respectively). As anticipated, all parental homodimers carrying a critical mutation in either the malonyl/acyltransferase (S581A) or β-ketoacyl synthase (K326A or C161A) domains lacked activity for either the malonyl/acyltransferase or the β-ketoacyl synthase partial reaction. Furthermore, heterodimers formed by hybridizing these mutants with either wild-type FAS or TE−FAS had half of the activity of the parental homodimer that exhibited normal activity. Two heterodimers consisting of one wild-type and one malonyl/acyltransferase mutant subunit were constructed with the His6- and FLAG tags on either of the two subunits; the two different heterodimers both exhibited half of the malonyl/acyltransferase activity characteristic of the wild-type FAS, indicating that neither the nature nor location of the tag affected activity of the dimers (Table I). Electrophoresis in SDS-polyacrylamide gels followed by Coomassie Blue staining revealed that all of the affinity-purified FASs were completely homogeneous, and Western analysis confirmed the presence of both His6- and FLAG-tagged subunits in all preparations (details not shown). These data confirmed that each of the highly purified heterodimeric species contained one subunit with a functionally normal malonyl/acyltransferase or β-ketoacyl synthase and one subunit with a defective malonyl/acyltransferase (MAT−) or β-ketoacyl synthase (KS−).

**Table I**

<table>
<thead>
<tr>
<th>FAS Dimers</th>
<th>Malonyl/acyltransferase activity</th>
<th>β-Ketoacyl synthase activity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>milliunits/mg % control</td>
<td>milliunits/mg % control</td>
</tr>
<tr>
<td>TE−/TE− (S2302A)</td>
<td>3310 ± 79 100 ± 2.4</td>
<td>177 ± 8 100 ± 4.5</td>
</tr>
<tr>
<td>TE−/TE− (S2302A), MAT−/MAT− (S581A)</td>
<td>0.5 ± 0.0 0.01 ± 0.001</td>
<td>93 ± 5 52 ± 5.4</td>
</tr>
<tr>
<td>TE−/TE− (S2302A)/TE−/TE− (S2302A), MAT−/MAT− (S581A)</td>
<td>1740 ± 25 52 ± 0.3</td>
<td>125 ± 14 100 ± 10</td>
</tr>
<tr>
<td>WT−/WT− (S581A)</td>
<td>3220 ± 60 100 ± 1.9</td>
<td>60 ± 11 49 ± 9.6</td>
</tr>
<tr>
<td>WT−/WT− (S581A)</td>
<td>1680 ± 100 52 ± 1.6</td>
<td>60 ± 10 49 ± 9.6</td>
</tr>
<tr>
<td>MAT−/MAT− (S581A)</td>
<td>3810 ± 105 100 ± 2.7</td>
<td>174 ± 8 100 ± 4.5</td>
</tr>
<tr>
<td>MAT−/MAT− (S581A)</td>
<td>10 ± 0.2 0.03 ± 0.005</td>
<td>60 ± 11 49 ± 9.6</td>
</tr>
<tr>
<td>WT−/MAT− (S581A)</td>
<td>1880 ± 120 49 ± 1.1</td>
<td>174 ± 8 100 ± 4.5</td>
</tr>
</tbody>
</table>

a Carboxyl-terminal FLAG-tagged.
b Carboxyl-terminal His6-tagged.
c Carboxyl-terminal His4-tagged.
d FLAG-tagged, within the noncatalytic region.

The stoichiometry of acyl chain assembly by heterodimers containing either one defective malonyl/acyltransferase or β-ketoacyl synthase domain per dimer.—The stoichiometry of acyl chain assembly was assessed by incubation of the FASs with [2-14C]malonyl-CoA, acetyl-CoA, and NADPH. As a control, we also prepared dimers containing the thioesterase mutation (TE−) in both subunits and heterodimers containing the ACP, thioesterase double mutation in one subunit and the thioesterase mutation in the companion subunit (ACP−, TE−/TE−); these FAS species were expected to assemble two and one long-chain acyl moieties per dimer, respectively. In preliminary experiments, the incorporation of radiolabeled malonyl moieties indicated that the stoichiometry of assembly of long-chain acyl moieties attached covalently to the FASs was less than anticipated (1.46 ± 0.04 fatty acyl chains/dimer). We suspected that FASs lacking a catalytically active thioesterase domain might synthesize and retain long-chain acyl moieties in the S9 cells before to isolation of the proteins, effectively blocking the 4′-phosphopantetheine from assembly of radioactive fatty acyl chains. Therefore, immediately before performing the affinity chromatographic steps, we treated the mutant FASs with rat mammary gland thioesterase II, an enzyme capable of removing acyl chains from thioester linkage to the 4′-phosphopantetheine of the ACP domain (24). Subsequently, the thioesterase was removed from the FAS preparations in the course of the affinity chromatographic procedures. Consistently higher incorporation of radiolabeled malonyl moieties was observed with these thioesterase-treated FASs (equivalent to 1.94 ± 0.14 long acyl chains/dimer). The control experiments confirmed that dimers containing defective thioesterase domains on both subunits (TE−/TE−) assembled two long-chain fatty acyl moieties, whereas dimers containing only one functional ACP domain and two defective thioesterase domains (ACP−, TE−/TE−) assembled only one long-chain fatty acyl moiety (Table III). The MAT−, TE−/TE− and KS−, TE−/TE− heterodimers both assembled two long-chain fatty acyl moieties per dimer (Table III). Identical results were obtained in parallel experiments in which the thioesterase activity was inhibited by treatment with phenylmethylsulfonil fluoride (15) rather than by mutation (details not shown). These results showed clearly that in dimers containing only a single active malonyl/acyltransferase or β-ketoacyl synthase domain, fatty acyl chains were assembled on the 4′-phosphopantetheine moieties of both ACP domains.

Catalytic Activity of Dimers Containing Only One Catalyti-
Heterodimers were formed by randomization of subunits from pairs of parental homodimers and isolated by dual column affinity chromatography. The FLAG and His$_6$ affinity tags were positioned at the carboxyl terminus (ct) or internally, within the noncatalytic region (id). The thioesterase mutation (TE$^\beta$) was S2302A; the ACP mutation was S2151A; the malonyl/acyltransferase mutation was S581A, and the β-ketoacyl synthase mutation was K326A. WT, wild type. Purified heterodimers (0.73 μM) were incubated at 37 °C for 30 s in 0.1 M potassium phosphate buffer, pH 6.8, containing 40 μM [2-14C]malonyl-CoA, 20 μM acetyl-CoA, and 110 μM NADPH, and the bound fatty acyl moieties were identified. Average carbon number of the product is equal to 1/100 × n(C×mol%), where C is the fatty acid carbon number, and n is the number of products formed. Data represent means ± S.D.s for three experiments.

### Table III
Stoichiometry of acyl chain assembly by heterodimers containing either one defective malonyl/acyltransferase or β-ketoacyl synthase domain/dimer

<table>
<thead>
<tr>
<th>Heterodimers</th>
<th>Total long-chain acyl moieties bound</th>
<th>Average length</th>
<th>Acyl chain length</th>
<th>FAS activity of heterodimers containing one normal and one mutated subunit</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mol/mol dimer</td>
<td>C atoms</td>
<td>mol %</td>
<td>FAS activity of heterodimers containing one normal and one mutated subunit</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>FLAG-tagged subunit</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>His$_6$-tagged subunit</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>milliunits/mg</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>% control</td>
</tr>
<tr>
<td>TE$^\alpha$, TE$^\beta$-ct-Flag</td>
<td>1.94 ± 0.14</td>
<td>19.4</td>
<td>1</td>
<td>WT$^\alpha$</td>
</tr>
<tr>
<td>ACP$^\alpha$, TE$^\beta$-ct-Flag</td>
<td>0.94 ± 0.06</td>
<td>19.3</td>
<td>1</td>
<td>WT$^\alpha$</td>
</tr>
<tr>
<td>MAT$^\alpha$, TE$^\beta$-id-His$_6$/TE$^\beta$-ct-Flag</td>
<td>1.96 ± 0.06</td>
<td>19.4</td>
<td>1</td>
<td>MAT$^\alpha$, (S581A)$^\beta$</td>
</tr>
<tr>
<td>KS$^\alpha$, TE$^\beta$-ct-His$_6$/TE$^\beta$-ct-Flag</td>
<td>1.60 ± 0.04</td>
<td>19.0</td>
<td>1</td>
<td>WT$^\alpha$</td>
</tr>
</tbody>
</table>

### Table IV

<table>
<thead>
<tr>
<th>FAS heterodimers</th>
<th>FAS activity</th>
<th>WT$^\alpha$</th>
<th>MAT$^\alpha$, (S581A)$^\beta$</th>
<th>WT$^\beta$</th>
<th>MAT$^\alpha$, (S581A)$^\beta$</th>
<th>KS$^\alpha$, (C161A)$^\beta$</th>
<th>WT$^\beta$</th>
<th>ACP, (S2151A)$^\beta$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>milliunits/mg</td>
<td>2260 ± 80</td>
<td>100 ± 3.5</td>
<td>1750 ± 54</td>
<td>77 ± 1.5</td>
<td>1078 ± 30</td>
<td>48 ± 1.4</td>
<td>1080 ± 14</td>
</tr>
</tbody>
</table>

$^\alpha$ The FLAG and His$_6$ affinity tags were positioned at the carboxyl terminus.

$^\beta$ The FLAG tag was positioned internally, within the noncatalytic region.

**Acknowledgment**—We thank Dr. Louis Liberti for preparing the decanoyl-S-pantetheine.

**REFERENCES**

Ketoacyl Synthase and Transferase Activities of FAS

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